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Cherished is the freedom symbolized by our holiday season. May the bells ring in good cheer and good fortune for you all through the New Year.

It is our fervent hope that those fighting far from home may soon return to their loved ones.

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 27 DECEMBER, 1944 NUMBER 12

OBSTRUCTION OF THE CENTRAL RETINAL VEIN*

A CLINICO-HISTOPATHOLOGIC ANALYSIS

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Chicago

During the past 40 years, the histopathology of obstruction of the central retinal vein has been discussed by many writers who have confined their publications to the histologic findings, of which they have presented a great and sufficient variety. The present study of a series of eyes lost in the course of venous occlusion introduces an attempt to correlate clinical with histopathologic data, making use especially of the newer knowledge, acquired during recent years, of the cardiovascular and hypertensive diseases, which often play an important role in the pathogenesis of the obstruction. The amount of pertinent histologic material in any laboratory is thereby limited, but such studies may yield eventually a better comprehension and clearer interpretation of the factors involved in the mechanism of venous occlusion, and more individualized and effective therapeutic methods.

The mechanisms of primary occlusion of the central vein already recognized from a combined clinical and histologic knowledge, can be classified into four types:

1. Occlusion by compression of the vein by structures external to it, through advancing sclerosis of the surrounding tissues, or through growth of tumor metastasis into the optic nerve, or slow expansion of benign tumors such as angiomas, within the optic nerve. This may occur either with or without secondary thrombus formation.

2. Occlusion by thrombosis secondary to inflammatory disease of the venous wall.

3. Occlusion by primary thrombus formation in blood dyscrasias.

4. Occlusion by the stagnation thrombosis following sudden reduction of the arterial-blood volume of the retina. From a purely histopathologic point of view the pathogenesis of only the first two types can be demonstrated beyond doubt.

The material presented here consists of two groups of eyes:

1. Twenty-four eyes which were lost because of secondary glaucoma consecutive to a primary obstruction of the central vein of the retina.

2. A small group of three eyes removed for various other indications, in which the histologic findings pointed to an imminent occlusion of the central vein. As the time elapsing between occlusion and enucleation is of decisive influence regarding the interpretation of primary essential and of secondary findings, the material of the first group is subdivided into:

(a) Eyes in which both occlusion of the central vein and onset of glaucoma were of recent origin, having occurred within six months of enucleation.

*From the Laboratory of Ophthalmic Pathology, Presbyterian Hospital. Read before the Chicago Ophthalmological Society, March 20, 1944.

(b) Those in which the obstruction of the vein occurred by more than six months to several years before the secondary glaucoma of recent development.

(c) Those in which occlusion as well as glaucoma are at least one year old. Groups b and c are excluded from the selection of case reports presented herewith, since the long time interval obscured the pathology of the primary occlusion. In many of the cases presented, there was a careful study of the fundus of the other eye at the time of the vascular accident, or of both fundi at a previous time and again at the time of enucleation.

The major portion of any histologic material dealing with venous occlusion consists of specimens illustrating the first and perhaps most frequent type of occlusion, that on the basis of sclerotic processes in and around the vein. In its essentials the histologic picture of this type of occlusion has been well defined by the investigations of Harms,[1] Verhoeff,[2] Scheerer,[3] and Coats.[4] The structures participating in this chronic sclerotic process are the central artery, the central vein itself, and central connective-tissue strand, and often glial remnants of the primitive epithelial papilla, which usually rest directly on the venous wall at or near its bifurcation, and which are known as the glial meniscus. The intralaminar portion of the vein has been demonstrated to be the invariable place of occlusion by such sclerotic processes.

The following series of illustrations will show, however, that the histologic picture in individual instances varies greatly with the predominance of sclerosis in one or the other of the aforementioned structures and with the coexistence of secondary features such as hemorrhagic imbibition, formation of a collateral circulation, and preretinal or prepapillary connective tissue. A knowledge of the clinical status of the patient is often of great value in accounting for the predominance of one or another of the primary or secondary findings.

Cross sections of the optic nerve were made in some of these eyes for the special purpose of studying the central vessels. In the others in which there were only the usual meridional sections, profiles of the central vein were made to illustrate its topography. These profiles, vertical or horizontal, depending upon the plane in which the bulb was sectioned, are made by drawing serial sections one over the other, the measurements in each section being obtained by use of an ocular micrometer after the method extensively used by Salzmann.[5]

Case reports

Case 1. M. M., aged 63 years, under observation for two years because of a mild diabetes and advanced essential hypertension (220/110), had a marked retinal arteriolar and arteriosclerosis together with an arteriosclerotic retinopathy which had reduced the visual acuity of each eye to 8/200. Especially noteworthy in the left eye was a slight narrowing of the retinal arterial vascular tree.

Some 15 weeks before its enucleation, a massive hemorrhage into the vitreous of the left eye obscured the fundus completely and reduced vision to bare light perception; within two weeks there was a rise of tension to 60 mm. Hg (Schiötz), which returned to normal only temporarily following an Elliot trephining operation. The clinical diagnosis was obstruction of the central vein of the retina, followed by secondary glaucoma.

The histologic examination of the posterior segment of the vertically sectioned bulb revealed moderate hemorrhagic imbibition of the retina in addition to the massive vitreous hemorrhage, and a large number of new-formed, wide, and thin-

walled preretinal veins and capillaries.* There were moderate retrodisplacement of the lamina cribrosa, more marked below than above, and considerable atrophy of the optic nerve.

The central artery was seen to be a wide, rigid tube filled with endothelial and subendothelial proliferation and some of the lamina cribrosa the vein became narrower, being encroached upon from below by a fibrous triangle which appeared to be part of the central connective-tissue strand. The superior wall of the vein was thickened throughout the intralaminar portion. Beginning at the junction of the superior and inferior

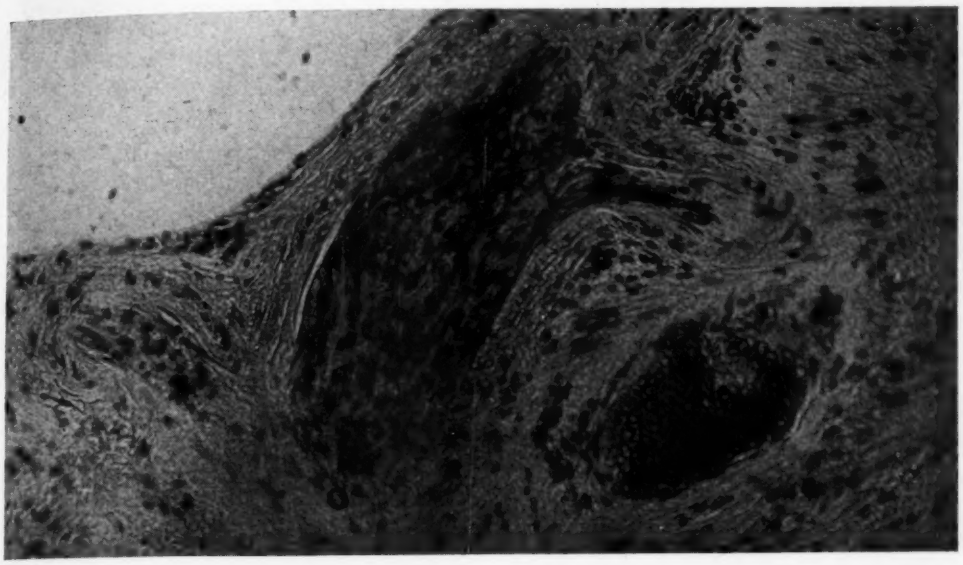

Fig. 1 (Klien). O, obliterating endarteritis, case 1.

elastic lamellation. These endarteritic lesions extended into some of the papillary branches (fig. 1). There was marked thickening of the media and adventitia of some of the retinal arterioles, but no hyalinization (fig. 2).

The histopathologic findings within the optic nerve and the central vein and its branches are illustrated in the vertical profile (fig. 3). There was a marked engorgement of the superior and inferior papillary veins, which united in the prelaminar region to form the central vein. A well-developed glial meniscus rested upon the bifurcation. Toward the middle papillary veins and extending backward to almost the posterior level of the lamina cribrosa the temporal and nasal walls of the central vein were connected by a solid mass which appeared to consist almost exclusively of endothelial cells. There was only a narrow column of blood at the level of this mass connecting the retrolaminar portion of the vein and the superior papillary vein. The narrow passage into the inferior papillary vein was completely blocked by a homogeneous coagulum having the appearance of a recent thrombus, secondary to the old and extensive endophlebitis. These intravenous lesions, which were densest and broadest at the temporal wall of the vein—that is, the wall opposite the artery—could be

* See illustrations in Klien, B. A. Retinitis proliferans. Amer. Jour. Ophth., 1938, v. 20, p. 434.

Fig. 2 (Klien). T, arterio-venous crossing, case 1; marked thickening of media and adventitia of artery. S, fibrosed central connective tissue strand; V, central vein with endothelial proliferations.

followed through seven consecutive sections, each 20 microns in thickness. Thus the horizontal diameter of the central vein within the lamina was only moderately reduced to about 140 microns, as compared to the normal 190-200 microns

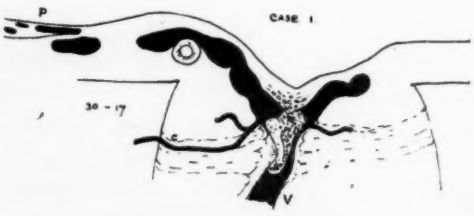

Fig. 3 (Klien). Vertical profile of central vein in case 1. P, retinitis proliferans; V, central vein; C, collaterals.

(Hertel[6]). A few small collateral venous branches could be traced through the anterior lamina toward the ciliary system.

The clinically observed narrowing of the retinal arterial vascular tree in this eye only, which was not spastic in character, pointed to reduction of the blood column in the central artery by sclerotic changes which formed the basis, after the venous occlusion, for the rapidly obliterating endarteritic process.

Case 2. S. M., a woman, aged 61 years, whose blood pressure was normal (140/94), had had severe diabetes mellitus for many years, but no ocular complaints until the sudden loss of vision of the right eye 12 weeks prior to its enucleation. The loss of vision was followed within a few days by severe pain in this eye and an increase of tension to 77 mm. Hg (Schiötz), which was not reduced below 50 mm. by miotics and a paracentesis. The right fundus was obscured by hemorrhage into the vitreous. The left eye was normal. The clinical diagnosis was secondary glaucoma following obstruction of the central retinal vein.

The histologic examination of the posterior segment of the horizontally sectioned bulb revealed an extensive vitreous hemorrhage, moderate hemorrhagic imbibition of the retina, and several large preretinal hemorrhages. There were several large, thin-walled preretinal veins directly connected with dilated veins of the

nasal portion of the retina. A plaque of prepapillary connective tissue also contained large new-formed vessels. There was considerable atrophy of the optic papilla but only slight retrodisplacement of the cribrous plate.

The lumen of the central artery (185 microns) was widely gaping, a sign usually pointing to rigidity of the arterial walls. The measurements of the thickness of the arterial wall were slightly increased (32 microns as compared to the normal 20 to 26), mainly due to a thickening of the adventitia. The latter and the central connective-tissue strand were poor in elastic fibers and well set apart from the surrounding tissues by increased fibrosis. Some of the retinal arteries showed a patchy fibrosis such as is often seen following healed atheromatous lesions, whereas other branches appeared normal

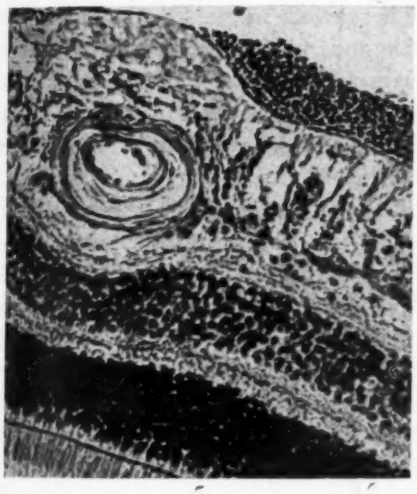

Fig. 4 (Klien). Atheromatous lesion in retinal vessel, case 2 (hematoxylin-eosin stain).

(fig. 4). The intralaminar space containing the central vein was considerably reduced, the venous walls were thickened throughout the intralaminar course and for a short stretch behind the lamina. A thick plaque of endothelial cells projected from the temporal wall of the vein, beginning at a sharp bend near the prelaminar junction of the papillary veins, leaving space only for a threadlike blood column along the nasal wall of the vein. From this plaque backward there extend-

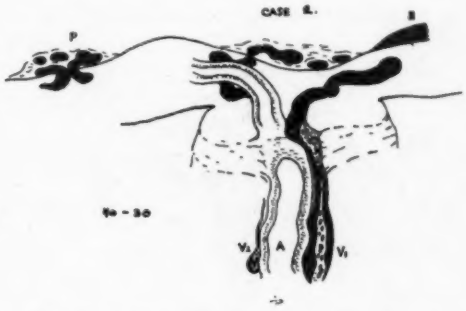

Fig. 5 (Klien). Horizontal profile of central vessels in case 2. A, central artery; V_1, central vein; H, preretinal hemorrhage; P, retinitis proliferans.

ed a band of varying width, also consisting mainly of endothelial cells, which crossed the free lumen of the vein and was attached to the opposite venous wall farther back (fig. 5).

Intravenous endothelial bands such as these are interpreted by some authors to be recanalization of a thrombus, but Verhoeff has shown that they are detached endothelium where small tributary veins have forced their blood behind degenerated endothelium, and thus formed a new channel in the manner of a dissecting aneurysm.

The essential features of cases 1 and 2 are similar. In both, the extensive formation of thin-walled wide preretinal collaterals pointed to impairment of the venous circulation for some time before the clinically diagnosed vascular accident. While in both, the intralaminar venous aperture was narrowed by a sclerotic artery, fibrosis of the central connective-tissue strand, and thickening of the venous walls, the main obstruction of the vein arose from endothelial proliferation anterior to this external encroachment upon

the vein. Hence this endothelial hyperplasia, which was most pronounced within the anterior lamina near the junction of the papillary veins, gradually tapering off within the posterior lamina, did not seem to be a secondary irritative proliferation due to venous collapse or excessive compression, but gave the impression of a coexistent phlebosclerosis on the basis of an endo- and mesophlebitis.

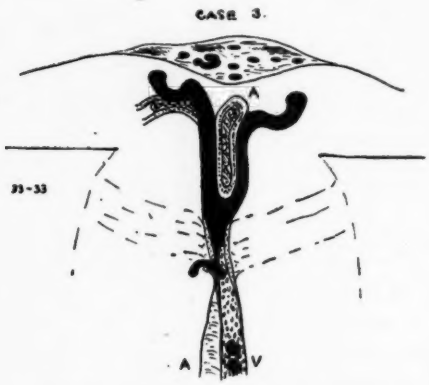

Fig. 6 (Klien). Vertical profile of central vein in case 3. A, V, as in figure 5.

The following two eyes present somewhat different aspects.

Case 3. B. R., a man, aged 54 years, had an essential hypertension (average blood pressure 170/110). Seven weeks prior to enucleation of the left eye, its vision suddenly was reduced to light perception, followed within a few days by pain and redness. The fundus was obscured by vitreous hemorrhage and the tension was elevated to 80 mm. Hg (Schiötz). After an Elliot trephining operation failed to reduce the tension, the eye was enucleated, on the basis of a clinical diagnosis of occlusion of the central retinal vein. The right eye was normal in every respect, with a vision of 1.5.

The histologic examination of the posterior segment of the vertically sectioned bulb revealed moderate diffuse vitreous hemorrhage and extensive preretinal and prepapillary hemorrhages in the process of organization into massive plaques of prepapillary connective tissue. There was still some edema of the optic nerve with incipient atrophy, and irregular, moderate retrodisplacement of the cribriform plate. The measurements of the central artery (CA) *in toto* were slightly increased in the intralaminar portion. The adventitia was normal, the muscularis was slightly, and the internal elastic membrane markedly, thickened and there was evidence of a longitudinal shrinkage of the artery, the elastica interna being markedly undulated and fixed in this position by smooth plaques of proliferated endothelium. Thus the lumen of the artery was considerably constricted. It was filled almost completely with endothelial and subendothelial proliferation which extended for a short way into the papillary branches. There was a marked thickening of the media and adventitia of all the retinal arteries and arterioles, but no hyalinization. The central connective-tissue strand (CCTS) was delicate, with an abundance of elastic fibers.

The central vein (CV) arose at the junction in the prelaminar region of the two papillary veins. Its walls were thick and rich in cells throughout the intralaminar course. Within the posterior half of the lamina the lumen was narrowed acutely by endothelial proliferation which seemed to fill it entirely at the posterior level of the lamina (fig. 6, vertical profile). Farther back an irregular lumen reappeared between the endothelial plaques. There was slight round-cell infiltration of the venous walls in the intra- and retrolaminar course. The horizontal diameter of the venous lumen within the posterior half of the lamina was even more reduced than the vertical, being about 80 microns; that is, less than half of the normal. Cross sections through the

optic nerve about 1 mm. behind the lamina still showed marked elastic lamellation of the internal elastic membrane of the artery, and a thickened muscularis. The venous lumen at this level was well filled with blood by tributaries from the septal system, but there was still an asymmetrical thickening of the endothelial and subendothelial layers.

Case 4. S. O., a man, aged 54 years, had a moderate arterial hypertension (158/100) of undetermined type. When first seen after one year of failing vision, the left eye had been painful for six months, was glaucomatous, with a tension of 71 mm. Hg (Schiötz), and had a beginning serpiginous ulcer.

Repeated paracentesis while the corneal ulcer was healing, and a later iridectomy, reduced the tension only temporarily. At the time of enucleation of the left eye, the tentative diagnosis was secondary glaucoma following a vascular accident. The right eye was normal in every respect, with a vision of 1.0.

Histologic examination of the posterior segment of the horizontally sectioned bulb showed marked hemorrhagic imbibition of the retina and infiltration with pigment-laden phagocytes. There was a slight retrodisplacement of the lamina cribrosa but no optic atrophy. Throughout the nasal half of the optic nerve there were convolutions of wide, well-filled veins, representing from all appearances an efficient collateral circulation whose place of exit from the optic nerve could not, however, be determined definitely, for the sections were at a right angle to the course of these vessels. In the prelaminar region there were also a number of capillary spaces which appeared to be just in the process of being opened up.

The total diameter of the intralaminar portion of the central artery was 240 microns; that is, only slightly more than normal. The outer part of its wall, adventitia plus media, was broader than normal, mostly owing to thickening of the adventitia, as shown by van Gieson stains. The entire intima was artificially detached and retracted, lying in the prelaminar lumen of the artery. The retinal arteries

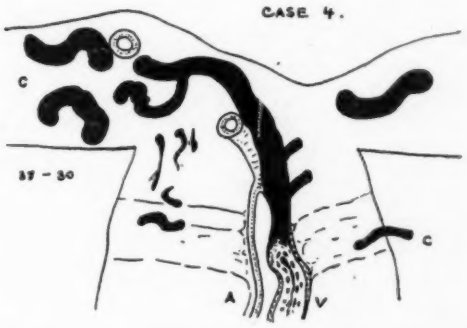

Fig. 7 (Klien). Horizontal profile of central vessels in case 4. C, collaterals.

appeared normal. The central connective-tissue strand was not thickened as a whole, but a cuneiform fibrous mass projected between central artery and vein from below.

The central vein, shown in horizontal profile (fig. 7), made a sharp bend at about the center of the lamina, where a considerable thickening of its walls began. From this place back to the cut surface of the nerve the lumen of the vein was completely filled with an old fibrous mass which was lined with endothelium and contained in its posterior part a slitlike blood column. Proximal to the sharp intralaminar bend the total vertical diameter of the vein was only about 65 microns, a third of the normal.

Remarkable in this case were the abundant wide collaterals, which coursed through normal nerve tissue, and therefore have to be considered as dilated preformed venous or capillary channels. Remarkable also was the absence of optic atrophy, considering that the duration of the secondary glaucoma was at least six

months. This finding supports the view that the degree of optic atrophy following an obstruction of the central vein depends upon the early efficiency of a collateral circulation, in the presence of which secondary glaucoma may exist for a long time without inducing extensive destruction of nervous elements. Lacking such

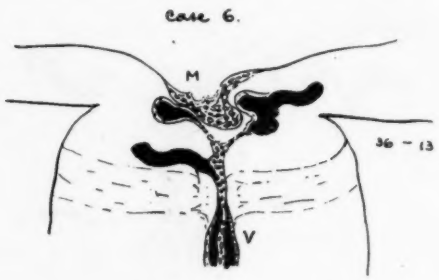

Fig. 8 (Klien). Vertical profile of central vein in case 6. M, glial meniscus.

collaterals optic atrophy seems to develop rapidly, as some of the cases presented here show, due to a combination of malnutrition and toxic action, to which only much later the destructive influence of increased intraocular pressure is added.

In both of these cases the pathologic findings responsible for the venous occlusion were limited to the posterior half of the lamina. In keeping with the younger age of the patients there was no fibrosis of the central connective-tissue strand. The vascular sclerosis dominated the picture with a markedly sclerotic artery encroaching upon the vein and a coexisting phlebosclerosis, which had led to considerable thickening of the adventitia and to occluding endothelial proliferation. As the lumen of the vein was considerably narrowed in both cases there is the possibility that some of the more recent endothelial proliferation was secondary to compression from which contact of the inner venous surfaces resulted. In case 4 an anomalous intralaminar course of the vein, and an anomalous configuration of the otherwise normal central connective-tissue strand associated with it, added to the embarrassment of the vein in this place.

Case 5. See Klien, "Anticoagulant therapy in occlusion of central vein of retina," Amer. Jour Ophth., 1943, v. 29, p. 701.

Case 6. F. M., a man, aged 71 years, suffering from mild arteriosclerotic hypertension (160/100; after therapy, 130/80), complained of overnight loss of vision of the right eye nine weeks prior to enucleation. The visual disturbance (bare light perception with faulty projection) was followed within four weeks by a rise of intraocular tension to 55 mm. Hg (Schiötz) which was reduced temporarily to normal by an Elliot trephining operation. Fundus details were never visible in the right eye. The left eye had a second-degree retinal angiosclerosis, and its visual acuity was 1.0.

Histologically there was a marked diffuse hemorrhagic imbibition of the retina, some edema of the optic nervehead with considerable rarefaction of the nerve fibers, and incipient retrodisplacement of the lamina cribrosa. There was a high degree of sclerosis of the CCTS and the central artery and its immediate branches, while many of the smaller retinal arteries appeared normal. A vertical profile (fig. 8) illustrates the findings in the papillary and central veins. The central vein had three papillary tributaries, two of which were collapsed before the junction. There was extreme narrowing of the central vein in its intralaminar course, and endothelial plaques and strands could be followed through it, beginning near the junction and ending a short stretch behind the lamina. The adventitia of the two partly collapsed papillary veins and the central vein was quite thick. The place of collapse of the veins seemed to be where they

coursed between the bifurcating sclerosed arterial branches. An unusually large glial meniscus penetrated deeply between these two venous branches. There were no visible collaterals.

In this case the picture of obstruction of the central vein was obviously produced by simultaneous branch occlusion of the superior and inferior papillary veins through marked sclerosis of the first branches of the central artery and an unfavorable topographic arrangement of these arteries and veins, although there was also extreme narrowing of the intralaminar central vein. A third papillary branch had remained patent and its blood stream had undermined the venous endothelium proximal to the collapse of the two other branches, in the nature of a dissecting aneurysm.

In the following two eyes which came from patients with malignant hypertension, the optic nerves are demonstrated in cross sections.

Case 7. C. B., a woman, aged 41 years, who had a diagnosis of early malignant hypertension, average blood pressure 235/125, had some intermittent obscurations followed by complete loss of the vision of the left eye six months prior to its enucleation. There were some hemorrhages into the vitreous and indistinctly visible large retinal extravasations. The onset of secondary glaucoma, with a tension of 90 mm. Hg (Schiötz), occurred four months before enucleation, and tension remained normal for several weeks after a trephining operation.

In the right fundus there was marked generalized spastic contraction of the entire retinal arterial vascular tree, and a marked venous displacement at the arteriovenous crossings. A few of the larger arterioles had irregularities of caliber which were thought to be possibly spastic as there was no visible retinal angiosclerosis. There was no retinopathy nor edema of the optic disc. Vision of the right eye was 1.5.

Cross sections through the optic nerve at the posterior level of the lamina cribrosa showed a normal CCTS, an oval

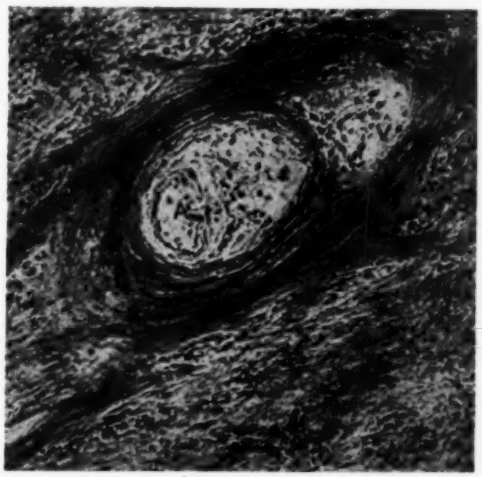

Fig. 9 (Klien). Cross section through central vessels in case 7 at posterior level of lamina cribosa (van Gieson stain).

cross section of the central artery, which exhibited marked thickening of the tunica intima. Muscularis and adventitia appeared normal. There was a triangular cross section of the central vein, the lumen of which appeared somewhat collapsed but not compressed and was extremely narrowed by subendothelial proliferation which arose mainly at the three pointed, collapsed angles of the triangular aperture (fig. 9). There was no appreciable thickening of the venous walls.

Cross sections through the anterior lamina cribrosa at the level of the venous junction showed extreme venous congestion but no endo- or mesophlebitis.

There was a marked thickening and hyalinization of the retinal arterioles (fig. 10).

Case 8. M. N., a woman, aged 56 years,

Fig. 10 (Klien). Hyaline degeneration of retinal artery, case 7 (van Gieson stain).

who had malignant hypertension (blood pressure varying from 300/140 to 240/92), suddenly lost the vision of the right eye six months prior to its enucleation. Obstruction of the central retinal vein was diagnosed, and after several months was followed by a rise of tension to 97 mm. Hg (Schiötz).

The fundus of the left eye showed excessive generalized narrowing of the retinal arteries and arterioles, and incipient arteriolar sclerosis.

Cross sections through the optic nerve at the posterior level of the lamina cribrosa (fig. 11) showed a normal CCTS and an almost collapsed central artery whose walls were only slightly thickened by hyperplasia of the elastica interna. The cross section of the central vein was oval and completely occluded by a fibrous plug. The lumen of the central vein remained collapsed throughout its retrolaminar course to the place of exit from the nerve 4.0 mm. behind the eyeball, and throughout this course there was considerable thickening of the walls by a subendothelial proliferation of connective tissue.

There was marked hyalinization of the retinal arterioles, the lumina of some of which were reduced to the diameter of an erythrocyte. Many of the larger retinal veins showed endothelial proliferation.

In both cases (7 and 8) there was a striking discrepancy in the amount of sclerosis of the CA and that of the retinal arterioles. Sclerosis of the CA consisted merely in hypertrophy of the elastica interna whereas the retina arterioles showed marked hyalinization. The central arteries did not show the rigid circular cross sections that were seen in the eyes of patients with late essential hypertension or arteriosclerotic disease, and thus did not encroach upon the central vein. In both cases the CCTS was normal.

The central veins were not compressed in their intralaminar course. It cannot be definitely decided at this stage whether the fibrous plug which completely occluded the venous lumen in case 8 and which contained also endothelial cells represents the end result of obliterating endophlebitis or the organization products of a thrombus. The marked pre- and retrolaminar phlebosclerosis and the incipient vascular sclerosis observed in addition to the spastic contraction in the

Fig. 11 (Klien). Cross section through central vessels in case 8 at posterior level of lamina cribrosa (Verhoeff's elastica stain).

fundus of the other eye seems to suggest a large element of primary phlebosclerosis in the pathogenesis of the venous occlusion in this eye. Case 7, on the other hand, presents the possibility of being the end result of a stagnation thrombosis, the requisites for which were given; that is, an early stage of a spastic type of hypertension, marked spastic contraction of the retinal arteries, and absence of advanced organic lesions as observed in the other eye. There was no pre- nor intralaminar phlebosclerosis.

The remaining cases of recent occlusion of the central vein are briefly as follows:

Case 9. C. H., a man, aged 32 years, had syphilis, left maxillary sinusitis, and normal blood pressure. Vision of the left eye was lost six months prior to enucleation, and there was a rise of intraocular pressure at some unknown time between the loss of vision and enucleation. Extensive retinal hemorrhages were dimly visible. The right eye was entirely normal. Histologic findings: Cross sections through the optic nerve in the anterior lamina cribrosa showed proliferating endophlebitis in one of the papillary veins. CA and retinal arteries were normal; retinal veins showed marked thickening of adventitia. The optic nerve had been cut too short for sections through the posterior lamina cribrosa.

Case 10. J. M. B., a man, aged 57 years, who had essential hypertension (blood pressure 200/105), lost the vision of the right eye five months before enucleation, secondary glaucoma following within four weeks. There was only a dull fundus reflex. The left eye was normal in every respect. Histologic findings: Proliferating endophlebitis in the central vein within the posterior lamina and retrolaminar course. The CCTS was normal; there was marked subendothelial and endothelial proliferation in the CA; thickening of the media and adventitia of the retinal arteries.

Case 11. X. H., a man, aged 72 years, two months prior to enucleation lost the vision of the left eye after two years of intermittent visual obscurations. Secondary glaucoma followed the loss of vision within a few days. The right eye was normal. Histologic findings: Occlusion of the CV by endothelial and subendothelial proliferation in the region of the posterior lamina, marked thickening of the CCTS, and adventitia of the CA. Retinal arteries were partly normal, partly showed fibrosis of walls as after atheroma.

Of the secondary findings in eyes with venous occlusion, the various types of collaterals are of special interest, as illustrated in the following cases:

Case 12. T. S., a woman, aged 68 years, who had an arteriosclerotic hypertension (blood pressure 180/100), suddenly lost the vision of the left eye two years prior to its enucleation. The horizontal profile of the optic nerve and central vessels (fig. 12) illustrates the complete obliteration of the CV through its intralaminar course and collaterals as follows: two on the temporal side, one of which formed a connection of papillary with choroidal veins or capillaries, the other with some of the veins in the subdural space. On the nasal side a wide vein took an extremely tortuous course through the lamina cribrosa and backward through the trunk of the optic nerve. It could not be determined whether it joined the central vein farther back or entered the subdural space.

The importance of more or less efficient collateral formation in the clinical

outcome of venous occlusion is illustrated in the three following clinical cases.

Case 13. X. C., a man, aged 46 years, who had arterial hypertension, cardiac hypertrophy, and insufficiency, albumen, hyaline and granular casts in the urine,

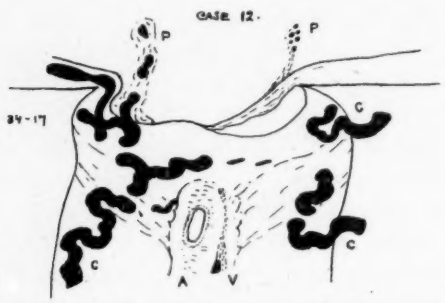

Fig. 12 (Klien). Horizontal profile of central vessels, case 12. C, collaterals.

gave the history of sudden bilateral diminution of vision eight years previously. Vision of the right eye recovered to 1.0, while that of the left eye remained poor: only 0.1, with a small central scotoma. The fundus of the left eye (fig. 13) revealed a normal optic disc, moderate generalized contraction of the retinal arterial vascular tree, and a striking course of some of the veins. Two large veins disappeared into the deeper layers near the superior margins of the optic disc after having been formed by confluence of tortuous malformed branches, one of them representing the inferior retinal vein. There was an area of faint pigmentary disturbance in the macula. The diagnosis of old, repaired occlusion of the central retinal vein was made. The history of bilateral sudden diminution of vision in a hypertensive patient is suggestive of a spastic origin, although no definite conclusion can be reached after the lapse of eight years.

Case 14. S. Z., a man, aged 49 years, who had essential hypertension (average blood pressure 177/118) and coronary disease, five years previously had noted painless failure of vision of the right eye to 10/200 with a central scotoma. The left eye had normal vision (1.2). The fundus of the right eye (fig. 14) showed a normal optic disc, a macular lesion consisting of ill-defined yellowish areas such as are often caused by glial retinal scars after extensive hemorrhages, and three anomalous venous channels, one in the upper half and two in the lower half of the optic nerve, of the appearance of opticociliary veins. The diagnosis of old, repaired obstruction of the central retinal vein was made.

An incidentally discovered anastomosis between retinal and choroidal veins is shown in fig. 15. It is the fundus of the right eye of a 64-year-old man with a myopia of 12 diopters and vision of 0.1 in this eye. Owing to the extensive peripapillary myopic atrophy of the pigment epithelium the place of transition of a very tortuous, perhaps new-formed, venous branch from the inferior retinal vein into a choroidal vein of extremely large caliber could be observed directly.

Fig. 13 (Klien). Fundus of left eye in case 13; eight-year-old repaired obstruction of central retinal vein.

There was an area of pigmentary disturbance in the macula.

It is impossible to decide in these cases which part of the collateral circulation consisted of preformed channels and which part of new-formed vessels. In each of them the collateral pathways appeared efficient. The visual disturbance appeared to be due to a macular lesion, which may have developed after extensive hemorrhages.

The following three cases are of special interest, inasmuch as they did not

Fig. 14 (Klien). Fundus of right eye in case 14. Five-year-old, repaired obstruction of central retinal vein.

present clinical manifestations of venous obstruction yet histologically showed conditions of the central vein almost identical with those found in cases of clinical occlusion, but less pronounced.

Case 15. E. C., a woman, aged 65 years, who had mild arteriosclerotic hypertension (blood pressure 154/88), and a moderate diabetes mellitus for many years, developed an acute glaucoma of the right eye three weeks prior to enucleation of this eye. There was an advanced rubeosis iridis of the right eye and an incipient

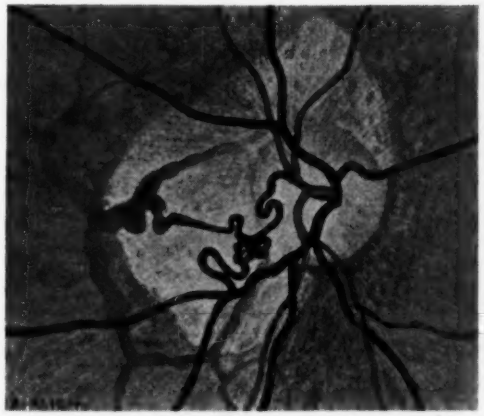

Fig. 15 (Klien). Clinically visible chorioretinal venous anastomosis.

rubeosis with normal intraocular pressure of the left eye. For the previous three years there had been gradual failure of vision of both eyes due to retinopathy.

Histologically there was moderate edema of the nervehead and an incipient rarefaction of the nerve fibers. The horizontal profile (fig. 16) summarizes the histopathologic findings in the central vein, which was moderately narrowed throughout its intralaminar course. Considerable thickening of its adventitia began within the posterior half of the lamina and extended for a short stretch backward. At

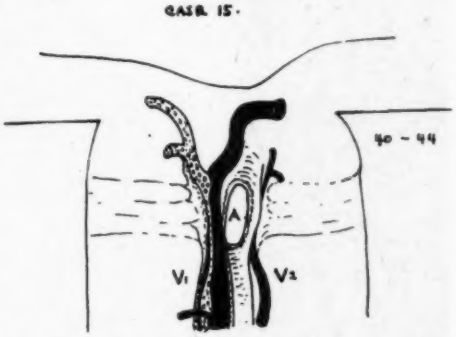

Fig. 16 (Klien). Horizontal profile of central vessels in case 15.

the prelaminar junction of the two papillary veins there was marked endothelial proliferation and bands of loose endo-

thelium could be followed through the intralaminar to the retrolaminar lumen of the vein, where they fused with the venous wall again. The temporal papil-

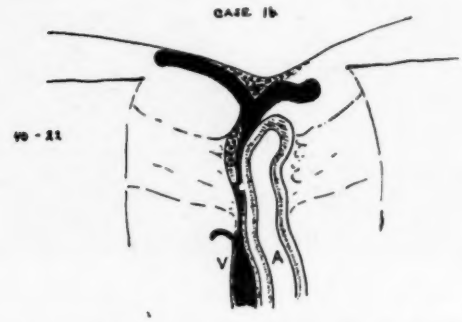

Fig. 17 (Klien). Horizontal profile of central vessels in case 16.

lary vein appeared occluded or collapsed but complete lack of massive retinal extravasations indicate that there was not an actually complete occlusion of this branch.

Case 16. J. K., a man, aged 70 years, who had a moderate arterial hypertension

Fig. 18 (Klien). Photomicrograph of central vessels in case 16 (hematoxylin-eosin stain).

of undetermined origin, had frequent attacks in the left eye of increased intraocular pressure due to subluxation of the lens. At the time of enucleation the tension was 80 mm. Hg (Schiötz) and, while the fundus was not clearly visible, there was no apparent retinal nor vascular pathologic change.

The histologic examination revealed an almost complete optic atrophy with some retrodisplacement of the lamina cribrosa. The central vessels are shown in the horizontal profile (fig. 17) and the microphotograph (fig. 18). There was extreme

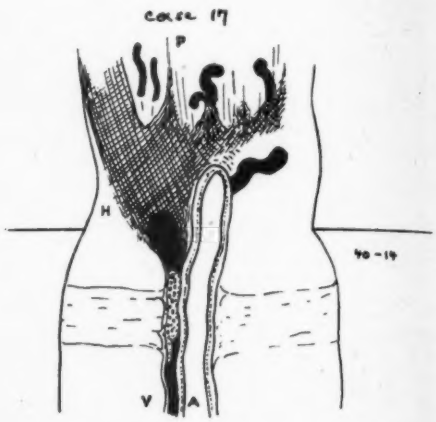

Fig. 19 (Klien). Horizontal profile of central vessels in case 17. H, hemorrhage; P, retinitis proliferans.

narrowing of the CV throughout its intralaminar course due to a fibrosed CCTS, marked sclerosis of the CA, and considerable thickening of the adventitia of the vein itself. Plaques of endothelial proliferation partly filled the venous lumen from the junction of the papillary veins backward through the anterior half of the lamina.

Case 17. T. F., a woman, aged 62 years, who had had arteriosclerotic hypertension (blood pressure 170/92) and diabetes mellitus of moderate degree for many years, had bilateral failing vision due to

an extensive retinopathy, retinitis proliferans, and, during the previous year, secondary retinal detachment. One week prior to enucleation of the left eye there was an acute rise of intraocular pressure which was not reduced by an Elliot trephining operation. This eye had an incipient rubeosis iridis.

The CV is pictured in a horizontal profile (fig. 19). While there was less narrowing of the lumen of the CV during its intralaminar course than in the two previous cases there was marked endothelial proliferation in the place which appears to be the site of predilection for it, judging from the frequency with which it occurred there in this series; that is, the anterior portion of the lamina cribrosa just proximal to the junction of the papillary veins. It is possible that the extensive retinitis proliferans was due to this type of venous disease.

Conclusions

1. Primary phlebosclerosis in the form of endo- and mesophlebitis plays a greater role than was heretofore supposed in the pathogenesis of venous occlusion, especially in those cases in which the occlusion is not due to senescence of the tissues alone, but to conditions accompanying systemic disorders.

2. The site of the most extensive proliferative endophlebitis is frequently the anterior lamina cribrosa or even the prelaminar region near the junction of the papillary veins, and is almost always associated with a marked narrowing of the vein in the posterior lamina due to mesophlebitis and encroachment upon the vein by sclerotic neighboring structures. Coexistence of these two findings suggests a partly mechanical origin of these endothelial proliferations, perhaps by vortex formations in the blood stream at the venous junction due to narrowing of the venous aperture farther back. A requisite for the ensuing endothelial proliferation would be a lack of normal resistance, as may happen in systemic diseases, and also in extreme senility.

3. Advanced age in patients without systemic disease and with normal blood pressure most often produces the well-known picture of obstruction in the posterior lamina, in which the final event consists in secondary, irritative endothelial proliferation due to extreme compression at the angles of a semilunar aperture.

4. In preëxisting arterial disease of the retina, venous occlusion and its consequences of stagnation, retrograde slowing of the blood stream, and the like, may bring about occlusion of the central artery in a relatively short time. The influence of venous occlusion upon the retinal arterial vascular tree, even without preëxisting arterial disease, can be demonstrated in the rare instances of intermittent venous occlusion in one eye, where it produces a unilateral retinal angiosclerosis in the eye with the repeated, even if short-lived, attacks of venous obstruction. It has been shown that an irritation of the inner surface of an artery or vein by a thrombus or by an endarteritic or endophlebitic process causes a reflex vasoconstriction of all the arteries in the corresponding area (R. Leriche[7]). Knowledge of this fact should be an added incentive to the prompt employment of therapy, especially the anticoagulants and antispasmodics.

5. The rapidity and efficiency with which collaterals develop, depend partly upon preëxisting channels, partly upon the rate at which the obstruction in the central vein develops. Thus in very gradual obstruction compensatory collaterals may be established before venous occlusion becomes complete.

6. Therapeutic suggestions derived from a clinico-anatomic study such as the

one presented here are naturally limited. The material upon which it is based consists *a priori* of the cases least amenable to therapy, as discussed in detail elsewhere (Klien[8]). It would seem, however, that in younger patients with such systemic disorders as hypertensive disease, diabetes, and syphilis the clinical picture of venous occlusion is often produced by a more widespread but less complete venous circulatory embarrassment than in older patients with senescence only; therefore, anticoagulant and antispasmodic therapy would seem to promise more favorable results in the former. But, even in the latter, prompt therapy directed against secondary thrombus formation, postponing complete obstruction of the vein as long as possible, may save the eye by giving the necessary time for development of efficient collaterals. The most promising cases are the ones with early spastic disease. Such cases as nos. 7 and 8 in this series have come to the histopathologic laboratory most likely because of the occurrence of the vascular accident prior to the advent of anticoagulant therapy.

References

[1] Harms, C. Anatomische Untersuchungen der Gafässerkrankungen im Gebiete der Arteria und Vena centralis retinae. Arch. f. Ophth., 1905, v. 61, p. 1.

[2] Verhoeff, F. H. Obstruction of the central retinal vein. Arch. of Ophth., 1907, v. 36, p. 1.

[3] Scheerer, R. Zur pathologischen Anatomie der Netzhaut-Zentralgefässe. Arch. f. Ophth., 1923, v. 112, p. 218.

[4] Coats, G. Complete obstruction of the central vein of the retina. Royal London Ophth. Hosp. Rep., 1904, v. 16, p. 262.

[5] Salzmann, M. Glaukom und Netzhautzirkulation. Abhandl. a. d. Augenh. u. i. Grenzg., Berlin, S. Karger, 1933, no. 15.

[6] Hertel, O. Beitrag zur Angiosclerose der Zentralgefässe des Auges. Arch. f. Ophth., 1901, v. 52, p. 191.

[7] Leriche, R. Quoted by E. Hartmann, Arch. of Ophth., 1942, v. 28, p. 605.

[8] Klien, B. Anticoagulant therapy of occlusion of central vein of retina, Arch. of Ophth., 1943, v. 29, p. 699.

BILATERAL METASTATIC CARCINOMA OF THE CHOROID WITH X-RAY THERAPY TO ONE EYE*

Report of a case

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San Francisco 8

Metastatic carcinoma of the choroid, while comparatively rare, has been reported a considerable number of times. Often it is bilateral, and in a definite percentage of cases the patient lives for one or more years after ocular metastasis occurs. By the time the eye is involved there is usually a generalized metastasis, so that any treatment to preserve vision is purely palliative. For obvious reasons the possible preservation of sight until death ensues is desirable. From the experience in the case reported here and in six other instances it would appear that radiation therapy offers some hope of accomplishing this end.

In 1872 Perls[1] reported the first metastatic tumor of the choroid in a man, aged 43 years, who had a primary carcinoma of the lung. In addition to the choroid of both eyes, many organs, including the brain, were involved. The subsequent cases have been collected and reviewed a number of times. Résumés of all the cases have been made by three authors. Usher[2] in 1923 collected 110 cases involving the choroid and iris; O. Ask[3] in 1934 collected 211 cases of uveal carcinoma in 59 of which the diagnosis was not histologically proved; in 1939 Giri[4] found that 170 cases of metastatic carcinoma of the choroid had been recorded. In addition to these mentioned, Lemoine and McLeod[5] reviewed all the cases of uveal metastasis and found 229 reports with 156 proved cases of metastatic carcinoma of the choroid on record.

Between the time of Giri's report and 1942 five additional proved cases have been reported by Schinz,[6] Kulvin,[7] Bonnet,[8] Von Sallmann,[9] and Pacheco.[10] In addition, two unproved cases were described by Aurand[11] and Luzsa.[12] More recently Bedell,[13] in presenting a photographic history of the development of bilateral carcinoma of the choroid in a woman aged 49 years, states that approximately 250 cases have been published or reported in discussion.

The incidence of metastasis to the choroid is low. As Ginsberg[14] pointed out Török and Wittelsdörfer found only one case in 366 cancers of the breast whereas Paquet found none in 733 cases. Schinz[6] within the last 20 years treated 536 cases of carcinoma of the breast at the Roentgen Institute of Zürich. Among these only three cases showed metastases into the choroid. Giri[4] believes that the rarity of choroidal metastases may be due to the fact that the ophthalmic artery branches from the internal carotid almost at right angles, so that cancer cells in the blood stream are easily swept past the comparatively narrow opening of the ophthalmic artery and deposited in the brain and meninges. It is Mann's[15] opinion that these metastases are probably much more common than is usually thought, as they are apt to occur in the terminal stages of carcinomatosis. Few ophthalmologists see more than one case; Fehr[16] saw four cases as did Greenwood;[17] Usher[18] observed five cases.

In by far the majority of cases the

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tumor is situated in the posterior pole of the eye. As Duke-Elder[19] states, one would expect the vast majority of emboli to travel up the 20-odd short posterior ciliary arteries rather than the 2 long posterior or the 5 anterior arteries. The posterior region of the choroid is therefore the site of election and the tumor is most frequently found on the temporal side near the macula where the short posterior arteries are most numerous and largest. According to Ginsberg[14] the left eye is more frequently involved than the right. Parsons[20] says this corresponds with the greater frequency of left cerebral embolism and is due to the same cause; namely, the more direct pathway by the left carotid which comes off from the aorta, whereas the right comes off from the innominate.

Involvement of the anterior segment is rare. Usher[2] in 1922 found only 8 cases, and Sanders[21] in 1938 found only 27, of which 17 were microscopically proved to be metastatic carcinomata. The choroidal metastases are more common than those of the iris and ciliary body by a ratio of 156 to 17. Sanders also feels that this can be explained by the numerical difference in the arterial distribution to the anterior and posterior uvea. Ask[3] pointed out that there are probably numerous cases of metastases to the ciliary body that are overlooked because they are small and cause no symptoms.

Involvement of the ciliary body alone is very rare, and only two cases have been reported. In 1929 Arnold Knapp[22] reported a metastasis in the ciliary body of a woman aged 68 years, who had had a breast removed for carcinoma eight years previously. The eye was enucleated and the woman lived 16 months longer. The second case was a patient of Ohr and Johnstone[23] who had an isolated metastasis to the ciliary body secondary to a carcinoma of the thyroid.

There have been only four cases in which the growth has been shown histologically to be limited to iris tissue. The first was reported by Proctor and Verhoeff[24] in a woman aged 72 years, with a carcinoma of the breast; the second was in a patient of Bolack, Bertillon, and Rogues[25] who had had a carcinoma of the breast removed three years previously; the third was in a man aged 68 years, with a carcinoma of the esophagus seen by Larson,[26] while in Sanders's[21] patient the site of the primary tumor was not located. In three of the cases the diagnosis was made following an iridectomy which included the carcinomatous nodule. In Larson's case this was done *post mortem.*

Smoleroff and Agatston[27] reported a case of metastasis to the retina without involvement of the choroid in a man of 55 years, suffering from an adenocarcinoma of the lower end of the esophagus and cardiac end of the stomach. This is the only recorded case of metastatic involvement of the retina by carcinoma.

The metastases are not infrequently bilateral, both eyes being affected, not, usually simultaneously but one following the other. Simultaneous involvement of the brain is common. Cohen[28] says that both eyes show metastases in one fourth of the cases but rarely simultaneously. Usher[2] gives the percentage of bilateral cases as one third whereas Lemoine and McLeod's[5] figures show 20.8 percent. Ginsberg[14] states that in 25 percent of the cases both eyes are involved and that the second eye may be involved by extension along the nerve. This, however, is not the rule, and the probabilities are that the metastasis in the second eye is due to a new hematogenous sowing not from the primary growth, but from the brain.

The site of the primary tumor in the majority of the cases is in the female breast. Cohen 28 gives 70 percent of the cases as due to mammary carcinoma,

while Usher,[2] who collected 90 cases, found 65 primary in the breast. Giri[4] in 1939 found that 65 percent of the cases were primary in the female breast. Giri also reported the case of a 69-year-old male who had a carcinoma of the breast with metastases to the choroid the metastatic tumor being typical of carcinoma of the breast. This is the only case of metastasis to the eye from a male-breast malignancy. Ewing[29] says that the frequency of carcinoma of the male breast varies between 0.86 percent of all mammary carcinoma in one series and 6 percent in another. In all, 244 carcinomas of the male breast have been reported. They occur in a later-age group than in women, are less malignant, persistent in growth, and may have local and general metastasis. Next in frequency to the breast, the lungs and bronchi, according to Giri,[4] account for 10 to 13 percent. Ask[3] found 15 percent of proved cases to be primary in the lungs and bronchi. Among the cases reported as secondary to malignancy in these structures were those of Ask,[3] Köpp,[30], and Koyanagi.[31] The alimentary tract accounts for about 7 percent of the cases. Among the more recent examples is one of the lower bowel reported by Kiep,[32] and one in the esophagus by Larson.[26] Only three histologically confirmed cases of metastasis to the choroid from a tumor of the rectum have been reported, those of Arisawa,[33] Dixon and Benedict,[34] and Von Sallmann.[9] The primary tumor has been reported in the prostate by Kulvin,[7] Greenwood and Southard,[35] Fehr,[16] Stallard,[36] and F. Ask.[37] Venco[38] reported a case originating in the thyroid. He was able to find five other cases recorded in the literature. They all showed a very slow course of metastasis. Choroidal metastases have also been reported from tumors in the liver and adrenals.[14] Holden and Rusk[39] report metastasis from a primary carcinoma of the ovary and Dimissianos[40] one from a carcinoma of the parotid.

It is rather generally accepted that metastasis takes place through the blood stream. Cohen[28] says the cancer cells are transported by the blood stream from the original sources to the ocular structures. Here the cells form emboli in the ocular vessels and finally break through the vessel walls into the surrounding tissues. This viewpoint is based on the finding of capillary- or precapillary-carcinoma emboli by a number of workers (Perls,[1] Ginsberg,[41] and Fry[42]). The lung finding would bear this out. Greenwood[17] reported four cases in which the lungs clinically or pathologically showed evidence of lung metastasis. O. Ask[3] points out that a positive lung finding makes a hematogenous metastasis probable; it has, however, been shown that cancer cells often appear in the lung capillaries without causing metastasis.

It can be statistically demonstrated that certain types of cancer of the breast, lung, and probably also prostate comparatively often produce uveal metastases, whereas other types such as cancer of the stomach, uterus, and ovary rarely affect the uvea. Thus while carcinoma of the breast causes over 50 percent of the uveal metastases, its general incidence is only 13.5 percent, while carcinoma of the stomach with a relative frequency of 36.5 percent gives rise to only 3.3 percent of uveal metastasis. O. Ask[3] says this can be explained on a basis that certain types of tumor have a tendency to hematogenous metastasis. He feels that the presumption of a specific affinity of the uveal tissue to certain ocular metastases does not seem indicated. Lemoine and McLeod[5] favor the theory of a specific affinity of uveal tissue, particularly the choroid, for cells coming from carcinoma of the breast.

Since 65 percent of the primary tumors

are in the breast, Giri[4] points out that 70 percent of the cases are in women, whereas in men, according to Usher,[2] the primary tumor is most frequently in the lungs. The greatest number occur in the age group between 40 and 49 years, the next group between 50 and 59 and the third between 30 and 39. Below the age of 30 and above 70 the occurrence is very rare.

The length of time between the appearance of the carcinoma and uveal metastasis varies a great deal. In carcinoma of the breast it may be a matter of weeks or years. In two cases reported by Rieth[43] the eye became involved 3 and 6 years, respectively, after amputation and in Kiep's[32] patient the choroidal lesion made its appearance 2½ years later. As already stated, Venco's[38] patient did not develop metastasis from a carcinoma of the thyroid until 14 years later. There was a similar length of time in the other five cases of metastasis from the thyroid reported in the literature. In Von Sallmann's[9] patient, who had a carcinoma of the rectum, there was a 10-year interval between the discovery of the primary tumor and the ocular metastasis. At times the metastatic growth in the choroid may be the first indication of malignancy. Reese[44] reported five cases wherein the metastatic lesion of the eye manifested itself before the primary site was located. Bietti[45] saw two patients with bilateral metastatic choroidal tumors in whom there was no evidence of a primary growth. Similar cases have been reported by Sanders,[21] Arisawa,[33] Dimissianos,[40] and many others.

Clinical course

The best descriptions of the clinical course are by Giri[4] and Fehr.[16] In the beginning one or more rather well-outlined round, pale-gray, pale-yellow, or yellowish-gray flat foci, over which the retinal vessels pass with a slight bend, appear at the posterior pole of the eye. The patch or patches increase in extent fairly rapidly and become slightly more prominent and, where they are close together, fuse into a large, flat, somewhat nodular tumor. At times there may be considerable mottling. Over the greater part of the tumor the retina is cloudy and opaque in appearance. This cloudiness disappears at the edges of the tumor which are not sharply defined.

The disc may become reddened, with blurred edges. While at first the retina lies on the surface of the tumor, sooner or later a folded detachment develops that becomes complete. The detachment seldom shrinks into a strand, as noted in choroidal sarcoma. As pointed out by Clapp,[46] on rare occasions in the further course of the carcinoma of the choroid the retina may become reattached to the tumor mass. The whole of the growth may spread rapidly in all directions and ultimately surround the disc. It always remains flat and is very rarely elevated more than 2 mm. There is no new vessel formation although small hemorrhages on the surface are not uncommon. In Köppl's[30] patient the picture was very suggestive of acute choroiditis. According to Usher[2] the tension of the eyeball may be normal, increased, or in a few cases be reduced. If the tension is increased it is usually amenable to miotics.

Certain characteristics help to differentiate choroidal carcinoma from sarcoma of the choroid. The increase of the growth occurs more rapidly than in a flat melanoma. Glaucoma is late in appearing, but pain is earlier and more pronounced than in primary tumors. In sarcoma, owing to the fact that the retina does not detach as early and does not become cloudy, the details of the tumor-surface formation are visible according to Fehr.[16] In carcinoma, on the other hand, the retina be-

comes cloudy and opaque early, so that the tumor is not visible or perhaps visible only at the edge. In carcinoma the lamina vitrea is rarely perforated but it can perforate, as does sarcoma, and grow into the subretinal space or perforate the retina. As the result of necrosis the tumor may cause inflammation; it may also extend along the nerve and may perforate the sclera. Bilateral involvement always suggests metastatic carcinoma, particularly in the presence of carcinomatous history.

Histologic picture

The typical pathologic changes in the ocular structures consist primarily in the presence of carcinomatous elements and have been described in detail by Ginsberg.[14] As already stated the metastases are found primarily in the area supplied by the short ciliary vessels of the posterior half of the globe. The cancer cells reach the small arterioles where they have been found by Abelsdorff,[47] Cohen,[28] and others. The cells seem to proliferate primarily in the small veins, however. The vessel is blocked, the cells proliferate and burst through the vessel wall to proliferate in the perivascular spaces and stroma. From here they can again break into other vessels. In the case described by Smoleroff and Agatston[27] the infiltrate around the vessels in the scleral canal was continuous with the mass within the eye. As previously stated, the tumor is almost always flat and not more than 2 mm. in thickness. Krukenberg[48] reported an unusual case in which the tumor filled two thirds of the eye. The lamina vitrea is rarely perforated, although Ginsberg[14] saw an instance in which the tumor had broken through into the subretinal space similar to the occurrence in sarcoma. Steichele[49] and others have shown that it is occasionally broken through from the disc.

With the development of the tumor the course may be exactly that of a primary choroidal growth in that it may cause detachment of the retina, glaucoma, and inflammation; can invade the optic nerve and sclera, and perforate externally. It is noteworthy that the choroidal carcinoma relatively frequently produces dissemination within the globe. Abelsdorff[47] and Ewing[50] among others have found small isolated nodules not connected with the main tumor, not only in the choroid but also in the ciliary body and iris. Orth[51] found the lamina cribrosa and anterior part of the nerve involved in this case. Evans's[52] patient developed metastasis to the optic nerve and chiasma late in the disease after the choroidal tumor had disappeared following radiation. While the sclera may be involved, extraocular extension is uncommon, probably because death usually occurs before this happens. It has, however, been reported by Usher,[2] Cohen,[28] and others.

According to Duke-Elder,[19] the histology of the tumor depends upon the nature of the primary growth, but varies considerably. In those tumors secondary to carcinoma of the breast the neoplasm consists, in most instances, of alveoli of various sizes containing large round polygonal cells with single large nuclei. Sometimes the stroma between the cells is scanty, in other instances it is profuse. The choroidal tissue is compressed and degenerates, and the chromatophores are disintegrated, which results in a dumping of escaped pigment. When the tumor is derived from tissue containing mucous membrane or glandular tissue (stomach, bowel, liver, and thyroid) or from the lungs it is adenocarcinomatous in type.

As has been stated, the type of tumor varies according to its primary source, and almost always it assumes the type of the primary cancer. Rather interesting is the case of Bock.[53] A primary tumor of

the liver had metastasized to the skin, muscles, arachnoid, lungs, orbit, and choroid. In the choroid there was a deep-green-colored, bean-sized tumor containing cylindrical cells arranged in tubules which contained green gall that chemically was shown to be biliverdin.

The rapid growth of the cells around the vessels may, at times, cause erosion and produce hemorrhage into the growth and surrounding area. The circulatory disturbance may also cause necrosis due to ischemia. As Uhthoff[54] pointed out, this necrosis may occur even in a relatively young tumor. Stock[55] reported a case of bilateral choroidal metastasis from a suprarenal capsule tumor in which the vessels were filled with tumor cells and the area surrounding these vessels showed considerable inflammatory reaction.

In addition to the typical metastatic carcinoma of the choroid from a primary lung tumor Koyanagi[31] found another probably benign growth had arizen from the pigment epithelium overlying the choroidal tumor.

Prognosis

The prognosis in these cases is invariably bad; they always end fatally.

In carcinoma of the breast the interval between the appearance of the tumor and metastasis may be weeks or years, according to Cohen.[28] In Rieth's[43] cases metastasis occurred three and six years after breast amputation. Kiep[32] had a patient with a carcinoma of the bowel in whom ocular metastasis did not appear for two years. In certain tumors there is an apparent tendency to metastasize late. In Von Sallmann's[9] case, a metastatic carcinoma of the rectum, there was a 10-year interval between the diagnosis of the primary tumor and ocular involvement. As Venco[38] has pointed out, the rare cases of metastasis from a carcinoma of the thyroid have all occurred years after discovery of the tumor, in his case 14 years.

Once metastasis has occurred the prognosis is extremely bad. Cohen[28] says that patients with unilateral cases may live a few years but that those with bilateral cases rarely live more than a few months. A great many of these patients are already cachectic, and multiple metastases are present, so that death may occur soon after the diagnosis of ocular metastases have been made, as was true of Köppl's[30] patient who died five weeks after the eye symptoms appeared. Usher[2] in 1923 stated that the average duration of life after the eye was affected was nearly eight months, the longest time recorded up to that time being two years. In 1938 Evans[52] reported a patient with bilateral metastasis who lived 2 years and 4 months after the diagnosis of ocular involvement had been made.

Treatment

It has been stated that treatment is palliative and unnecessary unless the eye is giving rise to pain. We have seen that some patients even with bilateral choroidal metastasis may live for a considerable time. Therefore any therapy that may preserve useful vision for the duration of life is justified, even though it is purely a palliative measure.

Therapy has been limited to irradiation, and there are only a few scattered reports of this form of treatment.

Wilmer[56] reports the case of a woman, aged 40 years, seen in October 1928, who five months prior to examination had had the left breast and axillary glands removed for adenocarcinoma. Three months later there were bilateral metastases, more marked in the left eye. The vision of the right eye was 20/20 and of the left, 20/200. The patient was given radium therapy of both eyes. A single portal of entry was employed on each side. A heavy lead cylinder with a 2-inch aperture was

placed against the skin of the temple and the radium placed in a cylinder 3 inches away from the skin. The beam was so directed as to "spare the cornea and perhaps the lens." Between October 15 and November 6, 1928, each eye received 16.8 gram hours of irradiation, and between January 10 and 16, 1929, an additional 12 gram hours were given.

Six months after irradiation was started the tumor mass in the right eye was chalky, yellowish-red in color, and was elevated only 3 diopters instead of 8 to 10. There were deposits of disintegrated and accumulated pigment granules in the retina over the tumor. In September, 1929, the right eye received 5.6 gram hours of radiation and the left 7.7 gram hours.

The patient died two years after irradiation had been started, and during this time the right eye retained normal function and the vision of the left eye remained at 20/200.

Zentmayer[57] in 1932 reported the case of a woman aged 60 years, who had bilateral metastatic carcinoma of the choroid secondary to a carcinoma of the right breast. The vision with correction was 6/60 in the right eye and 6/30 in the left. The tumors were the typical flat, yellow-white masses with some mottling of the surface. Radium treatment was administered in the form of two radium packs; felt 2 by 4 cm. and 1 cm. thick. Radium needles were distributed to give homogeneous irradiation over an area 2 by 4 cm. A total of 50 mg. was applied to each plaque and fixed to the right and left temporal regions just behind the external canthi. Filter of 1 mm. brass and 1 mm. lead was used; distance 12 mm. Further treatment was refused because patient's physician told her it was almost sure to produce glaucoma. There was a further gradual involvement of the choroid with vision finally being reduced to perception of hand movements. The tension never became elevated, being 18 mm. of mercury in each eye. Zentmayer also wrote that in a letter from Dr. deSchweinitz, he refers to a case of his own which was treated with radium at the Johns Hopkins Hospital, with beneficial results.

Evans[58] in 1937 described the successful use of radon in a bilateral choroidal metastasis secondary to a breast carcinoma. The patient, a woman, aged 41 years, had one eye enucleated three years after the breast amputation. Examination revealed a metastatic adenocarcinoma of the choroid. Six weeks later the right eye showed a metastatic tumor, with the vision reduced to 6/36. The eye was prepared as for diathermy operation and 4 radon seeds of 1.72 millicuries each were stitched to the sclera over the area of the growth. At the end of a week the radon seeds were removed by a second thread which had been attached to the seeds to permit removal. Two months later the vision was normal; the retina was entirely flat, the yellowish growth having disappeared. There was, however, a widespread choroidal reaction in all directions which involved the macular area. The field of vision was normal for 0.5-degree test object at 33 cm. Seven months later there was a recurrence of the growth above the disc. Radon seeds were again used in a similar manner. Two and a half months later the vision was 6/12 and the retina flat. Fifteen months later Evans[52] reported that the treatment had been effective for a period of 2 years and 4 months. During the last six months the vision failed owing to metastatic growths in the optic nerve and chiasma, as proved at autopsy. In his conclusion Evans stated that "radon may prove to be of value as a curative form of treatment in an otherwise hopeless condition and should be considered as an alternative to enucleation of the eye."

The use of X-ray therapy has been reported three times.

Uchermann[59] in 1928 reported the case of a woman, aged 35 years, who had an inoperable carcinoma of the breast. Clinically there was a bilateral metastatic carcinoma of the choroid. The patient was given three X-ray treatments of each eye over a period of four weeks. The vision of the right eye improved from 5/50 to 5/10 and the left from perception of fingers at 10 feet to 5/50, and the patient was again able to read. The fundi showed no demonstrable improvement. There was no further follow-up and no histopathologic report.

Lemoine and McLeod[5] reported on a 72-year-old woman with bilateral metastasis of a breast carcinoma in whom the left eye, which was enucleated because of pain, showed a metastatic carcinoma of the choroid. The right eye, which also contained a metastatic tumor, was given 1,645 r over a period of five weeks. Two months later there appeared to be about 1 diopter of elevation at the site of the tumor, with marked pigmentary changes about the macula and along the lower temporal artery. The vision was 15/50, and the patient was able to read and write. The lens opacity which was present before irradiation "had not changed much as the result of the radiation therapy." Microscopic examination of the right (irradiated) eye showed a flat tumor mass in the choroid with cell clumps showing the same appearance as those seen in the left eye. The majority of the tumor-cell nuclei were necrotic.

The third case was reported by Reese in the discussion of Lemoine and McLeod's paper. Additional data were obtained from Reese in a personal communication. The patient, a 53-year-old woman, reported to Dr. John Wheeler on March 29, 1936, complaining of eye symptoms of four months' duration. Right eye vision was the perception of fingers at one foot, left eye 20/200+ unimproved by lenses. Fundus examination: Right eye, media clear. Temporal to disc and extending well out into the periphery was a markedly elevated yellowish-gray area. There were numerous small hemorrhages upon this, and a densely pigmented area below and temporal to the macula. Along the upper edge there was a similar, less dense pigmentation. The lower portion of this area was connected by an isthmus to a grayish elevated area in the inferior portion of the fundus. Left eye: media clear. About and temporal to the disc, extending above and including the macula in its edge, was a raised yellowish-gray area. There were small yellowish and brownish areas upon this elevation. There was a large hemorrhage below and temporal to the disc. A diagnosis was made of metastatic carcinoma of both choroids, later found to be primary in the thyroid.

The right eye was enucleated, and microscopic study confirmed the diagnosis. X-ray therapy was directed to the left eye. Between March 3 and 8, 1936, the patient received 1,808 r of border-ray treatment. Between March 14 and June 24, 1936, 12 doses of X-ray irradiation, 300 r, were given to the left temple lateral to the canthus and 12 similar doses to the right side of the bridge of the nose. On June 11, 1936, the vision of the left eye with correction was 20/30. Two months later the lesion of the left eye seemed more extensive and the vision was 20/20—2. Two months later the patient died. Reese concluded that treatment by irradiation improved the vision apparently due to temporary regression of the tumor.

Case report

Mrs. D. B., aged 31 years, was referred by Dr. C. K. Mills of the Woodland Clinic for consultation on August 20,

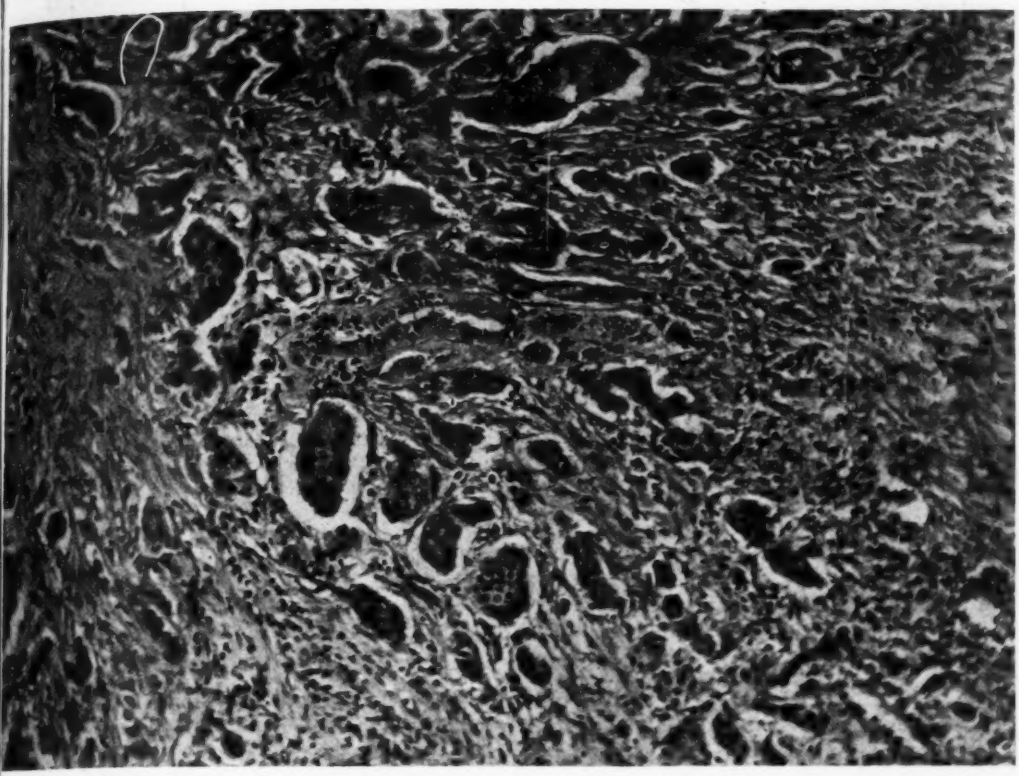


Fig. 1 (Cordes). Primary growth in breast. The cells are large and pleomorphic with occasional tendencies to form glands. A somewhat marked stromal reaction would suggest classification as a scirrhous adenocarcinoma.

1941, with a diagnosis of suspected metastatic carcinoma of the choroid. The patient had been seen for the first time two days previously and gave the history of poor vision in the left eye for approximately six weeks. Five months previously (March, 1941) the patient had had a radical amputation of the left breast for a carcinoma which had metastasized to the axillary lymph nodes. The radical amputation of the breast was performed by Dr. E. T. Rulison of Sacramento, who kindly forwarded the report from the pathology laboratory. From sections of the breast and axillary nodes a diagnosis of metastatic scirrhous carcinoma of the breast was made (fig. 1).

Examination. Vision, R.E., 1.0; L.E., 0.8. External examination was negative. Fundi, R.E., negative; L.E., media clear. A large detachment of the retina was present involving the nasal half of the retina and extending into the lower temporal quadrant. There was a large rather sharply outlined round area just nasal to the disc that was grayish-white in color, rather mottled in appearance, and solid looking. The retina appeared to be cloudy over this area but opaqueness was absent in other parts of the detachment. While the detachment was extensive it was rather flat (fig. 2).

Intraocular pressure, R.E., 19; L.E., 16.5 mm. Hg (Schiötz).

Transillumination was of no value.

Perimetric fields showed loss of field in the temporal half of the field extending into the upper nasal quadrant. This cor-

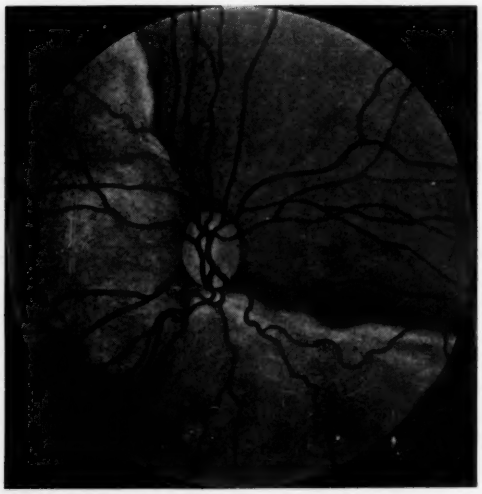


Fig. 2 (Cordes). Left eye. Showing large, rather flat detachment.

responded to the area of detachment. Perimetric field studies of the left eye were negative.

The diagnosis of metastatic carcinoma of the choroid was confirmed and, in view of the fact that there was no other involvement apparent on physical examination, enucleation was advised. Two days later the eye was enucleated by Dr. Mills, and the specimen was sent to Dr. D. Wood of Stanford University from whom sections were obtained.

PATHOLOGICAL REPORT (Dr. M. J. Hogan): The structures in the anterior globe are very distorted by the method of sectioning, but seem normal.

Choroid. There is a fairly extensive flat tumor of the choroid posteriorly in the region of the optic nerve (fig. 3). The cells are formed into strands by the choroidal stroma and are large and polygonal. A tendency to gland formation is seen here and there. There are three emissaria in the sclera beneath the tumor which are surrounded by tumor cells. At one point there are three small seedings into the retina over the tumor, but the subretinal space is only slightly involved. Only a few mitoses are seen. There is no keratinization (fig. 4).

Optic nerve cross sections: these show no extension of the tumor.

Diagnosis: Metastatic carcinoma of the choroid with extension into sclera and retina.

On September 17, 1941, four weeks after the initial examination, the patient returned complaining of flashes of light before the right eye.

Examination. Vision, R.E., 1.0. Fundus: Disc not remarkable. Just temporal to the macula was a flat detachment of the retina which was elevated 4 to 5 diop-

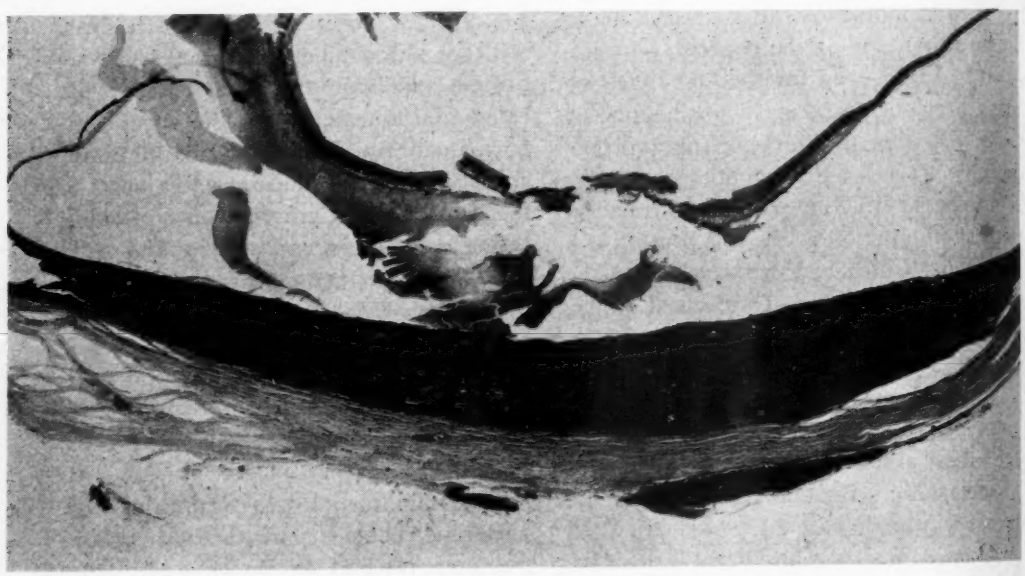


Fig. 3 (Cordes). Flat tumor of choroid posteriorly in the region of the optic nerve (×8).

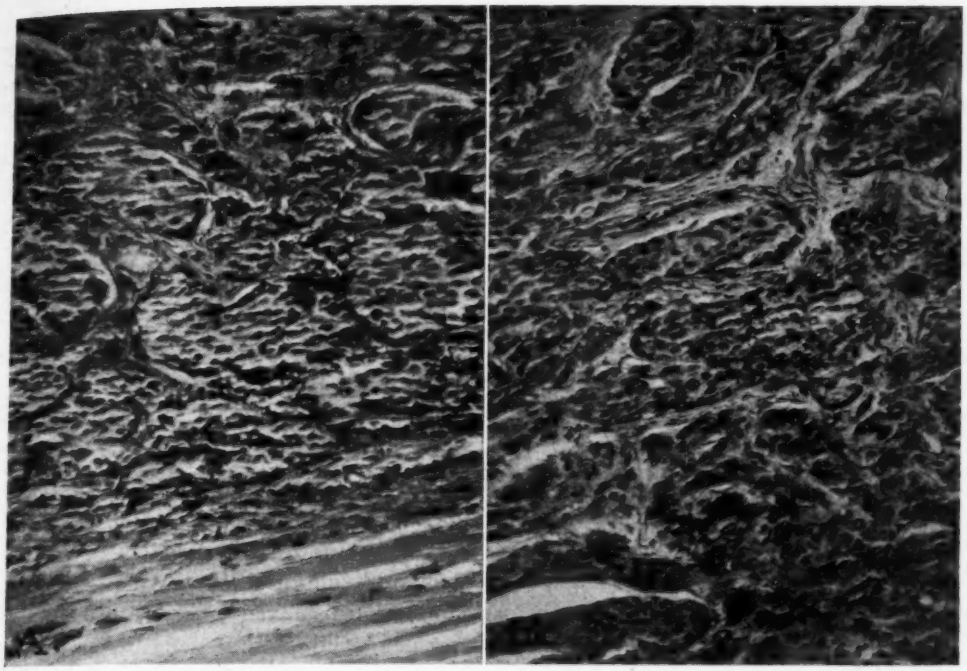
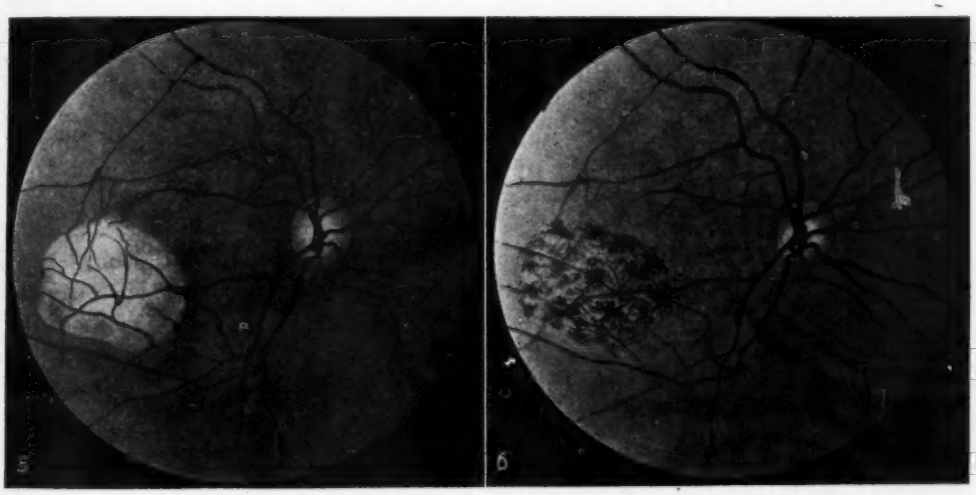


Fig. 4 (Cordes). Metastatic tumor (×120). The metastasis is composed of the same pleomorphic type cell arranged in strands and cords which have separated the choroidal lamellae in the region of the posterior pole of the eye. There is an occasional tendency to show gland formations. Stromal reaction is much less. The type is adenocarcinoma. A and B show sections from separate portions of the tumor.

ters. It was somewhat mottled in appearance and yellowish-gray in color. The area was sharply outlined and the detachment had a solid appearance.

Perimetric field examination revealed a scotoma in the nasal field corresponding in size and shape to the detachment of the retina (fig. 5).



Figs. 5 and 6 (Cordes). Figure 5, right eye showing the metastatic carcinoma of the choroid. Figure 6, fundus, right eye, after irradiation, showing pigment changes.

Tension 22 mm. Hg (Schiötz).

A diagnosis of metastatic carcinoma of the choroid was made and X-ray therapy advised.

*X-ray treatment.** On September 18, 1941, X-ray irradiation was started. The patient was treated from two lateral fields; the one on the right was rectangular 6 by 8 cm., and the one on the left was 10 cm. square. She received a total of 1,675 r to the right field and 1,640 r to the left. This was given over a period of one month. During the same time the patient was also given a roentgen castration with 1,000 r to two fields over her pelvis. The treatment factors were 195 K.V.P., 16 milliamperes, filter 1½ mm. copper and 1 mm. aluminum. The focal spot distance was 50 cm.

Dr. Mills wrote that 12 days after X-ray treatment was started the lesion was slightly but definitely less elevated and simultaneously with this decrease the patient stated that her annoying flashes of light had disappeared. One month after irradiation the elevation of the mass was about 1 diopter.

January 24, 1942, four months after irradiation, the patient was examined in the office.

Vision: R.E., 1.0. Perimetric fields and central fields showed no scotoma. In the fundus there was an area of retina corresponding to the site of the previously present tumor that seemed somewhat pale. The color seemed a little irregular, as though there had been some disturbance in the choriocapillaris layer and in the retinal-pigment layer. This was most pronounced above and temporal to the macula. The retina was flat.

The patient was seen at monthly intervals, and no change was observed until June 2, 1942, when she returned stating that she was having trouble with her legs due to involvement of the sacroiliac portion of the spine. She was receiving X-ray therapy for this lesion.

August 19, 1942. The patient complained of a return of flashes of light before her right eye. The vision remained at 1.0, and no visible change was seen in the fundus. Central fields on the screen did, however, reveal an indefinite defect in the nasal quadrant corresponding to the site of the lesion before X-ray therapy had been instituted. The lesion in the spine was better, and the patient had no trouble with her legs.

November 2, 1942. The indefinite field defect noted at the last examination had developed into a positive scotoma to the temporal side of the macula. Fundus examination showed a sharply circumscribed solid-looking detachment of the retina elevated almost 2 diopters in the area corresponding to the scotoma.

X-ray therapy was advised, and the patient received treatment from two lateral ports. "On the right side she was treated through a port 2 by 3 cm., centering on the posterior part of the orbit. The eyebrows and eyelashes were protected as far as possible with lead shielding. Through this port she was given 3,346 r measured in air at the skin. The other factors were 190 K.V.P., 16 milliamperes with filtration of 0.65 copper. On the left side a slightly larger port and heavier filtration were used. The area measured 3 by 4 cm. The same protection to the eyebrows was given, but no protection could be given to the eyelashes because of the enucleation. Through this port she was given 2,500 r but with heavier filtration. The other factors were the same. The filtration was 1.65 copper. Treatment was given daily over a period of a month and a half, treating alternating fields with the exception that the patient skipped one or two days and was not treated on Sundays."

* X-ray therapy was carried out at the Woodland Clinic by Drs. Earl Gray and Austin Clark from whom the data were obtained.

December 15, 1942. Vision R.E., 1.0. No scotoma present; retina flat—no further flashes of light.

February 12, 1943. Eyelashes previously lost were returning; otherwise condition remained unaltered. The patient had developed metastases in the lungs and shoulder girdle, for which she was receiving X-ray therapy.

April 26, 1943. Vision R.E., 1.0. The slitlamp failed to reveal any lens changes. Fundus: media clear, disc not remarkable. In the retina above and temporal to the fovea was an area that was lighter in color than the remainder of the fundus. Scattered through this area were fine dustlike clumpings of pigment. The retina was flat (fig. 6).

Perimetric fields showed a suggestion of a relative scotoma corresponding to the area of the retinal change.

Owing to progression of the lung lesions and development of new metastases in the lips, the patient was unable to return to San Francisco for further observation.

The patient died on July 16, 1943, 2 years and 4 months after the amputation of her breast and 23 months after the discovery of the tumor in her left eye. Dr. Clark of Woodland wrote that for two months prior to her death she had been having recurrent hydrothorax, bilateral, from pulmonary and pleural metastases. The vision remained normal throughout life.

The patient's mother refused autopsy and would not grant permission for removal of the eye.

Comments

Metastatic carcinoma of the uveal tract is rare; approximately 250 cases have been published or reported in discussions. Of this group 25 percent were bilateral.

The site of the primary tumor in the majority of cases is in the female breast. In addition the lungs, bronchi, alimentary tract, prostate, thyroid, liver, adrenals, ovary, parotid, and male breast have been noted as the location of the initial neoplasm.

The greatest number of cases occur in the age group of 40 to 45, the next group between 50 and 59 and the third between 30 and 39 years. Below this age and above 70 the condition is seen very rarely. The metastasis to the choroid may be the first indication of the malignancy.

The tumor has primarily the appearance of a flat, pale-pink, -gray, or -yellow area with a delimiting border that is faintly pigmented. Mottling is usually present. The tumor extends in surface rather than thickness, is thin, and is accompanied after a time by detachment of the retina; subsequent glaucoma may or may not accompany the lesion.

The prognosis in these cases, as has been pointed out, is invariably bad and they always end fatally. It has been said that patients with unilateral cases may live a few years, and that those with bilateral cases rarely live more than a few months. However, several cases have been reported where, with bilateral metastasis, the patient lived over two years. In one case the patient survived 23 months. While the prognosis is bad, any treatment that may preserve vision to the end of life should receive consideration.

Noteworthy also is that in the case here reported pulmonary metastases were present. Lemoine and McLeod found that 83 percent of autopsied cases showed pulmonary metastases.

From the few reports in the literature and personal experience it would appear that, in preserving useful vision, irradiation therapy is successful in a certain percentage of cases. The therapy has been given in two forms, radium emanations and X-ray irradiation.

The use of radium has been reported

by Wilmer, Zentmayer, and Evans. In Wilmer's case wherein one eye was seen early the preservation of 20/20 vision was possible and in the second more advanced eye the vision was 20/200. Evans was able to improve the vision from 20/200 to 20/20. This was retained for 2 years and 4 months. The application of the radon seeds must be done carefully. The eye is prepared as for a diathermy operation and the radon seeds are stitched to the sclera over the growth. It is then necessary later to remove the radon seeds, which can be done by means of a second thread which has been attached to the seeds to permit removal.

X-ray therapy has also been used successfully. The vision of Uchermann's patient improved from 20/200 to 20/40, that of Lemoine and McLeod's patient improved to 20/70, so that reading and writing were again possible. (In this patient a portion of the visual loss was due to lens opacities present before irradiation.) Reese was able to improve vision from 20/200 to 20/30 until a short time before death, when the vision dropped to 20/40−2. The patient herein reported retained 20/20 up to the time of death.

From the few available case records it is apparent that the earlier the patient is seen the greater the possibility of retaining useful vision.

The sensitivity of carcinoma to irradiation varies a great deal depending upon the type of tumor. This accounts for the variation of results obtained. In the case reported here the tumor, a primary scirrhous carcinoma of the breast, responded well to irradiation as was demonstrated by the rather easy control of the early isolated metastases to the spine, hip, and other parts. In both Wilmer's and Evans's patients the tumor was an adenocarcinoma of the breast.

As well as can be determined the results from radium and X ray are identical. The proper application of radium requires a rather formidable operative procedure; in addition, radium is not always available in a smaller community. X-ray therapy can be given by any well-trained roentgenologist and is accessible practically everywhere. Thus it would appear that X-ray therapy is the treatment of choice.

Recurrence may take place, as seen in Evans's case and in the one reported here. In both instances further irradiation caused the lesion to regress.

Of further interest is the tolerance of the eye to irradiation. The patient was given 3,315 r at the time of the initial irradiation and at the time of the recurrence, 14 months later, an additional 3,356 r were given, making a total of 6,671 r. Up to the time of death, eight months after the last irradiation, the lens failed to show signs of opacification. From this it would appear that if lens opacities develop it is not for some time.

Further experience with irradiation of these tumors may result in the indication for irradiation, even in monocular cases, before enucleation is considered, especially if the tumor is seen early.

Summary

1. A case is reported of a woman, aged 31 years, who had bilateral metastasis to the choroid secondary to a scirrhous carcinoma of the breast.

2. One eye was enucleated and the diagnosis confirmed by microscopic examination.

3. With the appearance of a similar growth in the second eye X-ray irradiation was employed. This was followed by disappearance of the tumor and retention of 20/20 vision in the eye. Fourteen months later there was a recurrence. The eye was again irradiated, with regression of the tumor.

4. Employing X-ray therapy to the eye made it possible for the patient to retain

20/20 vision up to the time of death (23 months).

5. While the ultimate prognosis is hopeless, the possibility of retaining useful vision by means of palliative irradiation should receive consideration in cases of metastatic carcinoma of the choroid.

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Bibliography

[1] Perls, M. Contributions to pathology of tumors. Virchow's Arch. f. Path. Anat., 1872, v. 56, p. 437.

[2] Usher, C. H. Cases of metastatic carcinoma of choroid and iris. Brit. Jour. Ophth., 1923, v. 7, p. 10.

[3] Ask, O. Metastatic uveal carcinoma. Acta Ophth., 1934, v. 12, p. 308.

[4] Giri, D. V. Metastatic carcinoma of the choroid secondary to mammary carcinoma in a man. Schweiz Med. Woch., 1939, v. 20, p. 1069.

[5] Lemoine, A. N., and McLeod, J. Bilateral metastatic carcinoma of the choroid—successful X-ray treatment of one eye. Arch. of Ophth., 1936, v. 16, Nov., p. 804.

[6] Schinz, H. R. Hematogenous distant metastasis in both eyes in carcinoma of the mammary gland. Klin. M. f. Augenh., 1939, v. 103, Oct., Nov., p. 425.

[7] Kulvin, Max. Metastatic carcinoma of choroid with primary focus in prostate. Amer. Jour. Ophth., 1940, v. 23, Aug., p. 892.

[8] Bonnet, P. Bilateral choroidal metastasis from cancer of the breast. Soc. d'Opht. de Paris, 1939, v. 51, Feb., p. 134.

[9] Von Sallmann, L. Gelatinous cancer of the choroid following carcinoma of the rectum. Arch. of Ophth., 1941, v. 25, Jan., p. 89.

[10] Pacheco, L. A case of bilateral metastatic carcinoma of the choroid. Guatemala Med., 1941, v. 6, March, p. 2.

[11] Aurand, L. Bilateral choroidal metastasis of a cancer of the breast. Bull. Soc. d'Opht. de Paris, 1939, v. 51, Feb., p. 136.

[12] Luzsa, A. Metastasis of carcinoma of mammary gland in both eyes. Klin. M. f. Augenh., 1940, v. 104, Oct., p. 495.

[13] Bedell, A. J. Bilateral metastatic carcinoma of the choroid. Arch. of Ophth., 1943, v. 30, July, p. 25.

[14] Ginsberg, S. Das Auge. In Henke and Lubarsch, Handbuch der speziellen pathologischen Anatomie und Histologie. Berlin, 1928, v. 11-1, p. 564.

[15] Mann, W. A. Discussions of Kulvin's paper. Amer. Jour. Ophth., 1940, v. 23, Dec., p. 1400.

[16] Fehr, O. Fundus picture in metastatic carcinoma of the choroid. Klin. M. f. Augenh., 1918, v. 60, p. 741.

[17] Greenwood, A. Carcinoma of the choroid. Trans. Internat. Congress of Ophth., April, 1922, Washington, D.C., page 571. Abst. Amer. Jour. Ophth., 1922, v. 5, Nov., p. 912.

[18] Usher, C. H. Frequency of metastatic carcinoma of the choroid. Brit. Jour. Ophth., 1926, v. 10, p. 180.

[19] Duke-Elder, W. S. Textbook of ophthalmology. St. Louis, C. V. Mosby Co., 1941, v. 3, p. 2523.

[20] Parsons, Herbert. The pathology of the eye. New York, G. P. Putnam's Sons, v. 2, p. 533.

[21] Sanders, T. E. Metastatic carcinoma of the iris. Amer. Jour. Ophth., 1938, v. 21, June, p. 646.

[22] Knapp, Arnold. Metastatic carcinoma of the ciliary body. Arch. of Ophth., 1929, v. 1, May, p. 604.

[23] Ohr, H. C., and Johnstone, I. L. Thyroid carcinoma with metastasis to ciliary body. Brit. Jour. Ophth., 1935, v. 19, Nov., p. 593.

[24] Proctor, F. J., and Verhoeff, F. H. A case of metastatic carcinoma of the iris. Arch. of Ophth., 1907, v. 36, p. 47.

[25] Bollack, J., Bertillon, F., and Rogues, A. Metastatic epithelioma of the iris. Ann. d'Ocul., 1927, v. 164, p. 336.

[26] Larson, V. Metastatic carcinoma of iris. Acta Ophth., 1933, v. 11, p. 386.

[27] Smoleroff, J. W., and Agatston, S. A. Metastatic carcinoma of the retina. Arch. of Ophth., 1934, v. 12, Sept., p. 359.

[28] Cohen, Martin. Bilateral metastatic carcinoma of the choroid. Arch. of Ophth., 1937, v. 18, Oct., p. 604.

[29] Ewing, James. Neoplastic diseases. Ed. 4, Philadelphia, W. B. Saunders Co., 1941, p. 593.

[30] Köppl, A. Multiple carcinomatous metastases in the eye. Klin. M. f. Augenh., 1935, v. 95, Sept., p. 310.

[31] Koyanagi, Y. Tumor-like growth of pigment epithelium next to a metastatic carcinoma of the choroid. Arch. f. Ophth., 1938, v. 139, Dec., p. 732.

[32] Kiep, W. H. Carcinoma of choroid secondary to carcinoma of breast and rectum. Trans. Ophth. Soc. United Kingdom, 1935, v. 55, p. 566.
[33] Arisawa, W. Metastatic tumor of the choroid. Klin. M. f. Augenh., 1914, v. 15, p. 695.
[34] Dixon, C. E., and Benedict, W. Adenocarcinoma of the sigmoid with metastasis to the eye. Amer. Jour. Surg., 1932, v. 15, p. 99.
[35] Greenwood, A., and Southard, E. Carcinoma of choroid metastatic from prostate. Boston Med. and Surg. Jour., 1903, v. 149, p. 286.
[36] Stallard, H. B. Cases of metastatic carcinoma of the choroid. Proc. Roy. Soc. Med., 1933, v. 26, p. 1042.
[37] Ask, F. Bilateral metastases of choroid from prostate. Hospitalstid., 1914, v. 7; quoted by Kulvin.[7]
[38] Venco, L. Choroidal metastasis of a thyroid tumor. Ann. di Ottal. e Clin. Ocul., 1935, v. 63, June, p. 401.
[39] Holden, W. A., and Rusk, G. Y. Metastatic carcinoma of choroid from primary growth in ovary. Arch. of Ophth., 1904, v. 33, p. 322.
[40] Dimissianos, B. Diagnostic difficulties in metastatic carcinoma of the eye. Klin. M. f. Augenh., 1932, v. 89, p. 356.
[41] Ginsberg, S. Das Auge. In Henke und Lubarsch, Handbuch der speziellen pathologischen Anatomie und Histologie. Berlin, 1928, v. 11-1, p. 562.
[42] Fry, W. E. Metastatic sarcoma of the choroid. Arch. of Ophth., 1933, v. 9, Feb., p. 248.
[43] Rieth, H. Two cases of metastasis to the choroid in carcinoma of the mammary gland. Klin. M. f. Augenh., 1936, v. 96, March, p. 369.
[44] Reese, A. B. Discussion of Lemoine and McLeod's paper. Trans. Amer. Ophth. Soc., 1936, v. 34, p. 149; also personal communications.
[45] Bietti, G. Prococious choroidal metastases of latent primary carcinoma and their diagnostic problems. Boll. d'Ocul., 1938, v. 17, March, p. 1638.
[46] Clapp, C. A. Discussions of Lemoine and McLeod's paper. Trans. Amer. Ophth. Soc., 1936, v. 34, p. 149.
[47] Abelsdorff, G. Bilateral metastatic carcinoma of the uveal tract. Arch. f. Augenh., 1896, v. 33, p. 34.
[48] Krukenberg, F. Metastatic carcinoma of the choroid. Klin. M. f. Augenh., 1903, v. 41, Beilageheft, p. 145.
[49] Steichele, H. Metastatic carcinoma of the choroid. Arch. f. Augenh., 1919, v. 84, p. 201.
[50] Ewing, A. E. Metastatic carcinoma of the choroid, ciliary body and iris. Arch. f. Ophth., 1890, v. 36, 1, p. 120.
[51] Orth, H. Metastases in choroid and optic nerve in mammary carcinoma. Klin. M. f. Augenh., 1935, v. 95, p. 625.
[52] Evans, P. J. Radon treatment of secondary carcinoma of choroid, post mortem observation. Brit. Jour. Ophth., 1938, v. 22, Dec., p. 739.
[53] Bock, E. A biliverdin containing tumor of the choroid. Virchow's Arch. f. path., Anat., 1883, v. 91, p. 142.
[54] Uhthoff, W. Metastatic carcinoma of the ciliary body. Deutsche Med. Woch., 1904, v. 30, 2, p. 1424
[55] Stock, W. Bilateral uveal metastases from suprarenal capsule tumor. Klin. M. f. Augenh. 1927, 78, Beilageheft, p. 116 (cited by Ginsberg,[14] p. 56).
[56] Wilmer, W. H. Atlas Fundis Oculi. New York, Macmillan Co., 1934, data accompanying plate 99.
[57] Zentmayer, W. A case of bilateral metastatic carcinoma of the choroid. Arch. of Ophth., 1932, v. 8, Oct., 616; also personal communication.
[58] Evans, P. J. The use of radon in the treatment of metastatic carcinoma of the choroid. Brit. Jour. Ophth. 1937, v. 21, Sept., p. 496.
[59] Uchermann, A. Carcinoma metastasis and x-ray treatment. Acta Ophth., 1928, v. 6, p. 170.

NOTES ON AN OPERATION FOR GLAUCOMA

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The operative procedure that is described in this communication was presented to the Indiana Academy of Ophthalmology and Otolaryngology as a preliminary report in April, 1937. At that time the operation had been performed upon 24 eyes (both eyes of six patients), beginning in January, 1934. The Academy members were informed that the operation was being presented to them in a spirit of humility engendered by the realization that it could probably be criticized for many reasons, and with the disturbing thought that it might have been previously outlined by some other eye surgeon. However, a rather careful study of the literature had not up to that time, and has not yet, uncovered the description of any operation just like it, although it incorporates some features of several other operations for the relief of glaucoma. The reason for calling it to the attention of the Indiana Academy was that it had given very satisfactory results with almost every eye upon which it had been performed. The fact that only a preliminary report was being made was definitely emphasized, since it was my feeling that a final report should not be made until at least 50 eyes had been subjected to the operation, and subsequently followed for at least two years, before reliable conclusions could be drawn regarding its efficacy. The description of the operation is as follows:

1. PREPARATION OF THE PATIENT

Is as for all other operations upon the globe, whenever it is possible to give sufficient time to the preparation. In the emergency of acute glaucoma, it is necessary to proceed with the surgery without the benefit of preliminary conjunctival cultures and so on.

2. ANESTHESIA

Five instillations of 4-percent cocaine solution are made at three-minute intervals, with 0.5 to 1 c.c. of 2-percent novocaine injected under the bulbar conjunctiva above the limbus. In acute glaucoma, injection of the ciliary-ganglion area with 1 to 1½ c.c. of 2-percent novocaine is required.

3. STEPS OF THE OPERATION

(a) After irrigation of the conjunctival sac and insertion of the speculum, a horizontal incision 12 mm. long and 1 cm. above the limbus is made in the bulbar conjunctiva with straight muscle scissors. The conjunctiva is dissected downward toward the limbus, the episcleral tissue also being picked up after the dissection has proceeded to a point below the insertion of the superior rectus muscle. Care is exercised to free the sclera carefully of all overlying tissue. The forceps, which are held in the operator's left hand, retain their grasp upon the conjunctival flap from the first snip of the scissors until the beginning of step (e). Fixation of the globe is thus maintained.

(b) With a Knapp knife needle, a horizontal scratch, 5 mm. long, is made in the sclera, 2 mm. above the cornea. This scratch is made with the knife-needle point, the stroke of the blade being made in the direction of its sharp edge, and with the knife held at an angle of about 45 degrees to the surface of the eye, instead of pointing straight in. After the initial stroke with the sharp edge of the point, thus definitely marking the line of the incision, the scleral section is completed deliberately and gently by strokes of the back, or dull side, of the knife-needle point. This step has for its objective the production of a purposely roughened, somewhat ragged incision in the sclera. As the last fibers of the sclera are divided, a quite characteristic sensation is transmitted to the surgeon's fingers, just as in the cyclodialysis operation. At this point great care and lightness of touch are indicated, to avoid injury to the underlying anterior portion of the ciliary body.

(c) A pair of fine-tipped, straight muscle scissors is next employed, to make a small snip at each end of the scleral wound. This snip is made with the scissor points directed toward the temporal limbus at the outer end of the incision, and toward the nasal limbus at the inner end. This procedure, which has been employed in the past at the inner and outer ends of a keratome incision for glaucoma iridectomy, has for its purpose the production of a flaplike valve. A filtering cicatrix, with bleb formation, is one of the objectives of this

operation. The purposely roughened scleral incision, with the snips at its ends probably aiding, *sometimes* accomplishes this objective.

(d) With the scleral section completed, a narrow iris spatula, somewhat curved, is introduced carefully into the incision. Hugging closely the inner surface of the sclera and cornea, the spatula is advanced into the anterior chamber. When its point comes into view in the anterior chamber, it is pushed gently forward to a position about half way between the pupillary margin and limbus. Danger of injury to the lens capsule is precluded by studiously avoiding the pupillary margin, thus keeping the iris between the spatula and lens. The spatula is next swept from side to side as far as the length of the incision will permit, thus separating the base of the iris from the cornea, and breaking up the meshwork of the iris angle. This step facilitates and makes safer the later introduction of the iris forceps for iridectomy. During the introduction and manipulation of the spatula, aqueous humor is drained from the anterior chamber, with the speed and amount of this drainage controlled by the care and precision with which the spatula is handled.

(e) The operator now transfers the conjunctival forceps to his assistant, who raises the conjunctival flap straight out from the globe, while a peripheral iridectomy is performed. The iris forceps are introduced with extreme care, their closed points hugging the inner surface of the sclera and cornea, just as the iris spatula was handled in step (d). This is the most difficult part of the operation, as the scleral opening is so small that it permits of only a moderate opening of the forceps. The latter are so flush on the surface of the iris, however, that only a very narrow opening of their tips is necessary to grasp the iris. Because of the location of the incision over the anterior portion of the ciliary body, it is probably reasonable to believe that a basal iridectomy is always obtained. Occasionally, in an eye that has rather markedly elevated tension, the iris will buckle into the wound as soon as the section is completed. In such instances the presenting iris is grasped, drawn out somewhat, and cut, with the deletion from the operation of steps (c) and (d).

The peripheral iridectomy may be replaced by the performance of a sphincter-dividing iridectomy or iridencleisis.

(f) The conjunctiva is closed with continuous or interrupted silk sutures, by picking up the subconjunctival tissue with the conjunctiva, and making sure that the former is drawn out smoothly over the underlying scleral incision.

The patient is kept in bed for two days and permitted to leave the hospital on the fourth or fifth postoperative day. No mydriatic is used unless there is evidence of uveal irritation. Ten days following the surgery gentle finger massage is begun for a short period three times daily. The patient is permitted to begin light work about two weeks after the surgery.

Between January, 1934, and the time of the original report in 1937, this operation had been performed upon 24 eyes, as was previously stated. Since 1937, approximately 125 additional eyes have been operated upon according to this method. Every type of glaucoma, including chronic noncongestive, subacute and acute congestive glaucoma, and elevated intraocular pressure following a cataract operation, has tested the procedure. It has been the author's experience that response to this operation has been at least as consistently satisfactory as to any other type of surgery. Several eyes have required more than one operation to bring the pressure down to a consistently low level. Such has been the experience of many surgeons who have used other types of glaucoma operations. The reasons for the need of more than one operation are becoming much more intelligible to us since the development of gonioscopy. This will be further discussed in a later paragraph.

The purposes of this surgical procedure may be outlined as follows: 1. A slow drainage of the aqueous humor through the gradually developed opening of the sclera by the scratch type of incision. 2. Development of a filtering cicatrix as a result of the purposely roughened scratch incision plus the extension of the incision toward the limbus by scissor snips. 3. The aid to drainage provided by what might be termed anterior cyclodialysis. 4. A consistently obtained basal iridectomy.

The reasons for the failure of a single operation, performed by this method, to bring the tension down and keep it down have become more apparent since some of the eyes that were operated on have been examined gonioscopically. Our local group has been making gonioscopic ex-

aminations for such a short time that it has been possible to examine only a few of our postoperative patients by this method. The things that we have seen have been most enlightening, and they may be outlined as follows:

1. The extent of our iridectomy has not represented a sufficient degree of chamber-angle circumference to bring the pressure down satisfactorily in some patients, because too-extensive peripheral anterior synechia made an iridectomy alone ineffective. (In such patients, a modification of the procedure to substitute iridencleisis would have given much better results.)

2. No evidence of a cyclodialysis opening or pit has been found in any chamber angle thus far seen.

3. No patients have as yet been examined gonioscopically for evidence of a communication from the chamber angle to a filtering cicatrix. As our number of operations has increased, the percentage of eyes which developed a filtering bleb following surgery has been sufficiently low that there is no sound reason to report a consistently obtained filtering scar by this operative method, although a good bleb sometimes results.

With the hoped-for advantages of a cyclodialysis opening and filtering cicatrix not consistently obtained by the performance of this procedure, we are given to wonder what if any advantage it offers. We believe that there are two advantages; namely, the slow leakage of aqueous and reduction of intraocular pressure obtained by means of the scratch incision, plus the basal iridectomy that is made possible by the location of the incision far enough back to come down upon the base of the iris with the iris forceps. Thus far, every postoperative eye that we have examined gonioscopically has exhibited a clean-cut basal iridectomy. There is no doubt that the scratch incision by invariably permitting slow drainage of the aqueous humor, is of the utmost value. Through this incision, a basal iridectomy can confidently be expected or an iridencleisis be performed with equal facility.

Gonioscopic examination will govern our preoperative plans in the future. Intelligent and rational choice of operation has been made possible by gonioscopy. The differentiation of wide-angle from narrow-angle glaucoma, together with determination of extent of angle block in the latter, will help us to decide whether cyclodialysis, iridencleisis, or basal iridectomy is indicated. Whatever type of operation we may elect, the advantage of the scratch incision remains constant.

In conclusion, it seems that the critical study of our results with this operation over a period of 10 years serves to prove the point that no operative procedure should be presented to our colleagues until a sufficient number of eyes has been subjected to the procedure and followed afterward for a sufficient time to give us an intelligent idea of the results.

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AMBLYOPIA IN CASES OF READING FAILURE*

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The frequent appearance of amblyopia among children referred for failure in learning to read prompted an investigation to find whether or not its incidence is really greater in such groups. The records of 100 children who failed to read were compared with those of an equal number known to be passing in reading but who had been referred for ocular complaints. The age range of the two groups was from 6 to 19 years, the average age of the poor readers being 10 years and that of the others 12 years. As is common in groups of reading-failure cases, previously studied by others as well as myself, 80 percent of the poor readers were boys. The group that had no reading trouble was found to be evenly divided between boys and girls.

The criterion of amblyopia was visual acuity of less than 20/20 in either or both eyes without demonstrable lesion, but unimproved by lenses. Each macula was examined with particular care, but no lesions were seen here nor elsewhere in the fundi of any eyes in either group. The poor readers exhibited twice as great a frequency of ambyopia of all categories, slightly less than twice as frequent bilateral amblyopia, and four times as frequent amblyopia of the left eye only. No cases of amblyopia in the right eye only were found in either group. A little less than three times as many poor readers were *equally* amblyopic in each eye. The

TABLE 1

COMPARISON OF DATA FROM CASES OF READING FAILURE WITH THOSE FROM SUCCESSFUL READERS

	Cases of reading failure	Successful readers
Number of cases	100	100
Age range in years	6 to 19	6 to 19
Average age in years	10	12
FREQUENCY (% of each group)		
Amblyopia in either or both eyes	25	12
Amblyopia in both eyes	21	11
Amblyopia in left eye only	4	1
Amblyopia in right eye only	0	0
Both eyes equally amblyopic	17	6
Both eyes amblyopic, left the more so	3	2
Both eyes amblyopic, right the more so	0	3
Better vision with both eyes than with either separately	2	1

CENTRAL TENDENCY

	Right eyes	Left eyes	Both eyes	Right eyes	Left eyes	Both eyes
Average (arithmetic mean) visual acuity of amblyopic cases (Snellen)	20/40	20/60	20/30	20/40	20/50	20/30

* From the School of Education, Boston University.

incidence of bilateral amblyopia with a greater degree of defect in the left eye was only slightly different in the two groups, whereas the poor readers presented no case in which the amblyopia was greater in the right eye than in the left. Twice as many poor readers experienced better vision binocularly than with either eye separately. A quarter of the same group were amblyopic in one or another category and about a fifth of them were amblyopic in both eyes.

The average amount of amblyopia was studied in terms of visual acuity below 20/20. The only difference between the groups in this respect was that the poor readers showed an average of one Snellen line poorer vision (that is, greater amblyopia) in the left eye. The average visual acuity for the right eyes only and both eyes together was the same in each group.

Amblyopia may be either a neurologic defect, concomitant with partial word blindness, or a primary factor impairing reading ability through visual inefficiency. It is probably one of many physical factors that participate to a greater or less degree in different cases of reading failure. The fact that the incidence and average amount of amblyopia of the left eye only was greater among poor readers discloses an unexpected tendency which will bear investigation in later studies.

The treatment of children who fail to read and are amblyopic involves the usual remedial teaching techniques supplemented by sightsaving-class methods, when vision is very poor, and by amblyopic exercises. Not much is to be expected of such exercises, but they should be tried. Sometimes surprising visual gains follow, especially among children in the lower grades. This may be due to late maturation. If so, amblyopic exercises may foster the process of visual development.

Summary

The records of 100 cases of reading failure were compared with those of an equal number of children who were known to be passing in their school studies. A considerably greater incidence of amblyopia was found among the poor readers, and they exhibited a greater average amount of amblyopia in the left eye only. The average amount of amblyopia in the right eye only and in both eyes was the same in each group.

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ANGIODIATHERMY OF THE LONG POSTERIOR CILIARY ARTERIES AND ITS USE IN THE TREATMENT OF GLAUCOMA*

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In 1890 Wagenmann[1] carried out his classical experiments in which he studied the effects of cutting the ciliary arteries of rabbits. Among other things, he found that a marked hypotony which persisted for several days developed when only one long posterior ciliary artery was cut. Since the tonometer had not been perfected at that time, he made no effort to carry out tension studies nor did he suggest that such a procedure might have any practical value. The present study, therefore, was undertaken to compare the results following electrocoagulation of the long posterior ciliary vessels with those of Wagenmann and to learn whether or not such an operation can be applied to the problem of glaucoma in man.

Comparative anatomy of the long posterior ciliary arteries of man and rabbit

In man the long posterior ciliary arteries arise from the ophthalmic artery, enter the sclera obliquely to either side and slightly above the optic nerve, and after pursuing an intrascleral course of from 3 to 7 mm. enter the suprachoroidal space, where they course forward in the horizontal meridian to the ora serrata (Fuchs[2]). Here they bifurcate, pass forward alongside the ciliary muscle to its anterior border, and then proceed inward to form the greater arterial circle (Leber[3]). The ring is completed by anastomotic vessels which course between the arms of the bifurcation. In rare instances the vessels course through the muscle substance, and at such times can be found between the meridional and circular fibers, but in the majority of cases the vessels pass not through the substance of the muscle but externally alongside of it (Maggiore[4]).†

From the greater arterial circle vessels are given off to the iris, ciliary body, ciliary processes, and anterior choroid. In addition to the long posterior ciliary arteries, the greater arterial circle receives as tributaries the anterior ciliary arteries, which pass through the sclera in the region of the rectus-muscle insertions.

The long posterior ciliary arteries of the rabbit have a somewhat different origin from that of man. The temporal vessel arises from the external ophthalmic artery, a branch of the internal maxillary artery, while the nasal artery arises in part from the external ophthalmic artery but also receives a small twig from the internal ophthalmic artery (Davis,[6] Krause[7]). Both arteries run slightly below the horizontal meridian and enter the sclera about 2 mm. posterior to the equator. Here they run superficially until they turn deeply inward to disappear some 2 mm. from the limbus. After disappearing from view each vessel bifurcates, giving rise to a superior and inferior branch which form the greater arterial circle. From this vessel, as in man, branches are

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† Fortin[5] teaches that the converse holds true—that is, that in the majority of cases the vessels pass through the muscle—but his teachings are at variance with those of other anatomists.

given off to supply the iris, ciliary processes, and anterior choroid (Baurmann[8]). Thus it can be seen that the blood supply of the anterior uveal tract is similar in man and rabbit, and experiments carried out on the long posterior ciliary arteries of the rabbit should be applicable to man.

Methods

In all the experiments on rabbits except those in which cyclodiathermy was carried out, monopolar desiccating current was used. This was simply a matter of expediency, since by so doing the necessity of shaving the animals for application of the neutral electrode was eliminated. The current used was of such strength as to give a spark gap of about 1 mm. The Schiötz tonometer was employed in all tonometric determinations. Due to the fact that the corneal curvature of the rabbit is not the same as that of man and since the footplate of the instrument is made to conform to the curvature of the human cornea, a valid objection to the use of this instrument can be raised where absolute readings are desired. In these experiments this argument cannot be brought to bear, since, for the most part, tonometric readings of fellow eyes are compared, and tonometric determinations, at best, only approximate the true intraocular pressure. In those instances wherein a simple tension curve was plotted, the difference in tonometric readings are too large to be attributed to tonometric discrepancies. Before deciding upon the tonometer to be used, the Souder and Schiötz instruments were carefully compared. In our hands the Souder tonometer proved most inconsistent and it was given up in favor of the Schiötz instrument.

All operations on animals were carried out under nembutal anesthesia given intraperitoneally, 0.045 gm. of nembutal per kilo of body weight being used routinely. It was employed in a solution of 10-percent alcohol, in which each cubic centimeter contained 0.045 gm. of the drug. Where this proved insufficient, supplementary injections of the solution were made in the marginal ear vein until the desired state of anesthesia was reached. A 0.5-percent solution of pontocaine was instilled into the conjunctival sac. All operations were performed with the animal's head held immobile in a clamp. Sterile instruments were used, but no preoperative care was given the eye, nor was the animal draped. When tonometric readings were taken other than at the time of operation, 0.5-percent solution of pontocaine was used locally, the animal was mummified by wrapping in a towel and held firmly by an assistant so that the cornea was parallel to the floor.

Coagulation of a single long posterior ciliary artery

A single long posterior ciliary artery of one eye was coagulated in each of six rabbits, the fellow eye being left untreated to serve as a control. Tonometric readings, observation with the corneal microscope, corneal sensitivity, and fundus studies were then carried out at appropriate intervals.

The conjunctiva was incised about 5 mm. from the limbus in the horizontal meridian (either anteriorly or posteriorly, depending upon whether the nasal or temporal vessel was to be treated). At first a speculum was employed, but it was found more expedient to have the lids retracted by the assistant with finger traction. Tenon's capsule was incised, and the anterior lip of Tenon's firmly grasped with Elschnig forceps. The artery could be identified at once, but in some instances before coagulation could be carried out the overlying rectus muscle had to be pushed aside with a strabismus hook. An Arruga detachment speculum was then introduced to obtain better exposure. After the vessel had been identified, coagulation with the desiccating current was carried out just anterior to the entrance of the vessel into the sclera and for a distance of 2 or 3 mm. A curved-needle electrode was used. Treatment was considered adequate when the blood column was interrupted and the treatment area turned a deep-gray color. Closure of the conjunctival wound was effected with a running silk suture in some of the first cases but later this was abandoned and the conjunctiva simply replaced. Rapid healing resulted in both instances (fig. 1).

Results. Tension was taken immediately before operation and at intervals postoperatively. Figure 2 shows a characteristic curve which illustrates the rapid fall in tension to about one half the preoperative level over a period of three hours.

Tension curves of control and treated eyes of the six animals are shown in figure 3. From these curves it is evident that the low tension continues for several days and in most instances is lowest between the second and fourth days postoperatively, after which it rises slowly to attain the preoperative level in about two weeks. In one instance (animal no. 213) the tension never fell to an unusually low level. Coagulation of either nasal or temporal vessel gave similar tension curves.

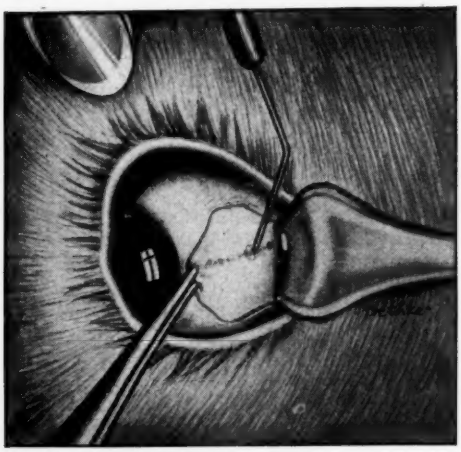

Fig. 1 (Guerry). Angiodiathermy in the rabbit.

On the first postoperative day, slight edema of the corneal epithelium was noted by means of the corneal microscope, it being more pronounced in animal no. 208. Dilation of the peripheral ring vessel was also present. By the third day no pathologic changes were present other than slight fullness of the iris ring vessel, which persisted in three cases for more than two weeks. At no time was a positive Tyndall effect present nor were cells seen.

Corneal sensitivity was found to be definitely impaired in the quadrant adjacent to the treated area in three animals. This was determined by touching the cornea methodically with a firm bristle but it was felt that the results obtained were inconclusive.

The postoperative changes observed with the direct ophthalmoscope differ somewhat in the pigmented and albinotic animals owing to the fact that in the pigmented animal the choroid is obscured by the pigment epithelium. For this reason the changes in the two types of animals are described separately.

In the albino, immediately after operation a whitish area some four or five disc diameters in size can be seen in the region where diathermy was carried out. At first a few partially filled choroidal vessels are present in the blanched area but these disappear leaving the area dead white. Fanning out in wedge shape anteriorly as far as can be seen is a blurred, striated area of yellowish hue. No change takes place until 72 hours have elapsed, when the treated area appears covered with a fine grayish net and the wedge-shaped area begins to regress. An occasional vessel can be seen at the periphery of the dead-white area after four days. After a week a small portion of the wedge-shaped area has disappeared and after two weeks only a small portion remains. After one month the area treated by diathermy appears as a small grayish-white avascular spot the size of which is dependent upon the area actually treated with diathermy.

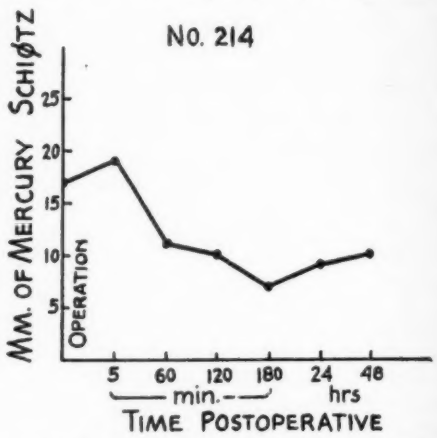

Fig. 2 (Guerry). Immediate postoperative tension fall following coagulation of a single artery.

In the pigmented animal a similar process is noted except for the fact that no choroidal changes can be observed. Pigment deposition begins around the periphery of the treated area after one week. The peripheral sector disappears after three weeks. Pigmentation of the treated area continues until eventually the lesion shows changes which vary from a mild granular pigmentary disturbance to that of a large area showing destruction of choroid and retina and associated with dense pigmentation. The anterior region usually shows slight pigmentary stippling. At no time was there any evidence that the opaque zone was elevated.

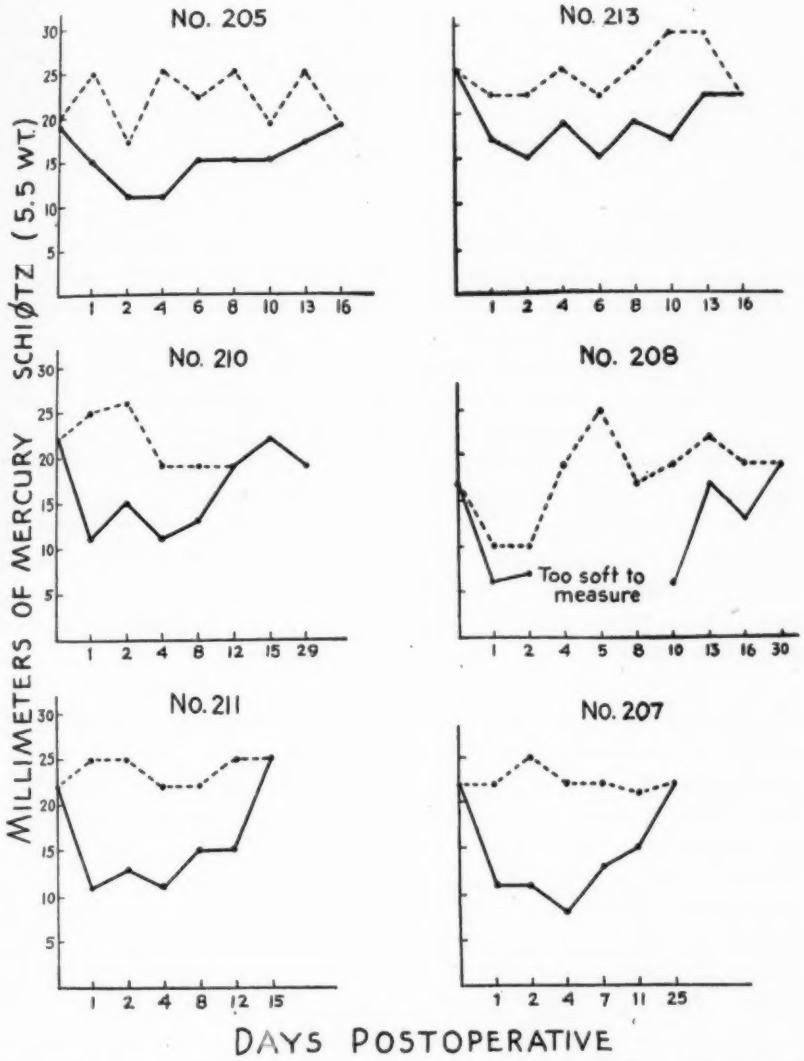

Fig. 3 (Guerry). Tension curve of control eyes and of eyes operated on following coagulation of a single artery. Broken lines represent control eyes, and solid lines treated eyes.

Discussion. The corneal edema noted is due to the slight damage to the endothelial cells. In animal no. 208 the endothelium evidently suffered more than that

of the other animals. Slight decrease in the corneal sensitivity in the region adjacent to the treatment area should be expected, since the long ciliary nerve supplying that area runs close to the artery and in sections is found constantly involved with the artery in the eschar. A more sensitive method of testing might reveal decreased sensitivity in all cases. No attempt was made to evaluate the possible effects which might have been due to the destruction of motor fibers coursing in the long ciliary nerve, but it was felt that any such effect must be minimal.

The changes seen with the ophthalmoscope in the treated area are due to an actual coagulation of choroidal and retinal elements followed later by necrosis with complete destruction of architecture and finally fibrosis. Anteriorly a disintegration of the neuroepithelial elements associated with edema takes place, giving rise to the ophthalmoscopic picture noted. This change is apparently caused by the interruption of the blood flow through the anterior choroidal vessels.

Wagenmann first tenotomized the tendon of either the internal or external rectus muscle, depending upon which artery was to be cut, placed an enucleation spoon behind the globe, which was pulled forward and rotated until the ciliary vessels could be seen, and then cut the long ciliary vessel, which presented, with scissors. Hypotony developed and was thought to be lowest shortly after operation. There was no change in corneal sensitivity. The fundus changes noted resembled those seen following angiodiathermy in the region anterior to the treatment zone. In his cases, however, at least one half of the retina was involved, the region being confined to that side on which the artery had been severed.

The explanation for the more extensive retinal involvement in Wagenmann's animals must be attributed to the different techniques employed. It is not unlikely that at the time the long posterior artery was cut some of the short ciliary vessels might also have been injured. Since in the rabbit these arteries supply the posterior part of the choroid the overlying retina would be affected in that region as well as anteriorly.

Coagulation of both long posterior ciliary arteries

Both long posterior ciliary arteries in each eye of six rabbits were coagulated by the same technique employed before and the same studies were carried out.

Results. In all cases an immediate tension drop similar to that shown in figure 2 was noted. Five eyes, in which the cornea remained grossly clear the day following operation, followed a course comparable to those in which only one vessel was coagulated. In two eyes, wherein the corneas showed slight gross clouding 24 hours postoperatively, the tension curve followed that of rabbit no. 208 of figure 3. In the remaining five eyes, the corneas of which showed gross clouding 24 hours after operation, the tension was too low to register and remained so, the eyes becoming phthisical after a time.

In the five eyes wherein the corneas were grossly clear, slight corneal edema was present for two days postoperatively. A slight Tyndall effect was also present for one week and rapidly moving cells were noted for a period of four or five days. No synechiae formed, and iris injection was limited to slight fullness of the peripheral ring vessel.

Considerably more corneal edema, which persisted for five or six days, was noted in the two eyes showing slight gross clouding. These eyes also showed a moderate Tyndall effect and slight but definite deep stromal clouding in the nasal and temporal sectors. Numerous rapidly

moving cells were present. The stromal clouding disappeared after 4 to 6 days and the Tyndall effect after 10 to 12 days.

Twenty-four hours after operation there was marked corneal edema and dense opacification of the entire thickness of the stroma in the nasal and temporal sectors of the five eyes in which pronounced corneal clouding had been noted. This corneal clouding spread centrally from the nasal and temporal sectors where it began about 10 hours after operation. With a fine slit the cornea appeared to be twice as thick as normal, the thickening being most pronounced peripherally. The Tyndall effect was marked, and in the albino animals, numerous slow-moving cells were present, while the pigmented animals showed in addition large pigment flecks on the endothelial surface as well as fine pigment granules dispersed throughout the aqueous. Occasional posterior synechiae were present. After 48 hours no detail could be made out because the opacification of the cornea had progressed until it had become uniformly opaque. Thickening had increased until now the cornea was at least three times its normal thickness. These changes resembled those seen in parenchymtous keratitis of man and resembled this condition even more strikingly when, on the fifth to sixth day, numerous fine brushlike vessels began to invade the stroma from the limbus. These vessels progressed until after two or three weeks only a small central area, about 5 mm. in diameter, remained vessel free. At this stage the cornea assumed a striking beef-red color, the center alone remaining milky white. After this peak vascularization had occurred, the vessels began to regress and the clouding to disappear. After two months the vessels had disappeared to gross inspection and the cornea had cleared considerably. The globe during this time became markedly shrunken and through the yellowish tinted, moderately clear cornea, the anterior chamber was seen to be absent, the iris atrophic, and the lens cataractous. The corneal changes are shown in the series of pictures contained in figure 4.

In these animals an attempt was also made to study the corneal sensitivity. In two cases presenting clear corneas, sensitivity was perhaps slightly impaired in the nasal and temporal sectors, and in one instance perhaps in the nasal region. Sensitivity was definitely reduced throughout the entire cornea in all of the corneas which became opaque. The length of time this diminution in sensitivity persisted is not known, but in one instance after a year, when the globe had become completely phthisical, corneal sensitivity appeared to be normal.

The fundus changes observed followed a course similar to those noted when only one vessel was coagulated. Corneal clouding precluded fundus examination in some of the cases, but even in these instances, as long as an examination could be made, the process was found to be essentially the same.

Discussion. In those instances wherein the cornea remained grossly clear and the tension curve followed the pattern seen after coagulation of a single artery, the changes brought about by operation must still be minimal. The fact that a Tyndall effect, along with cells in the aqueous, was present indicates that tissue damage was somewhat more pronounced in those eyes than in those wherein only the one vessel was coagulated. Why these eyes should remain relatively unhurt by a procedure which in other eyes results in extreme tissue destruction and phthisis, is best explained in one of two ways: either they possess a singularly extensive collateral blood supply to the greater arterial circle, or, and this is more probable, only a partial or temporary obliteration of one of the long posterior ciliary

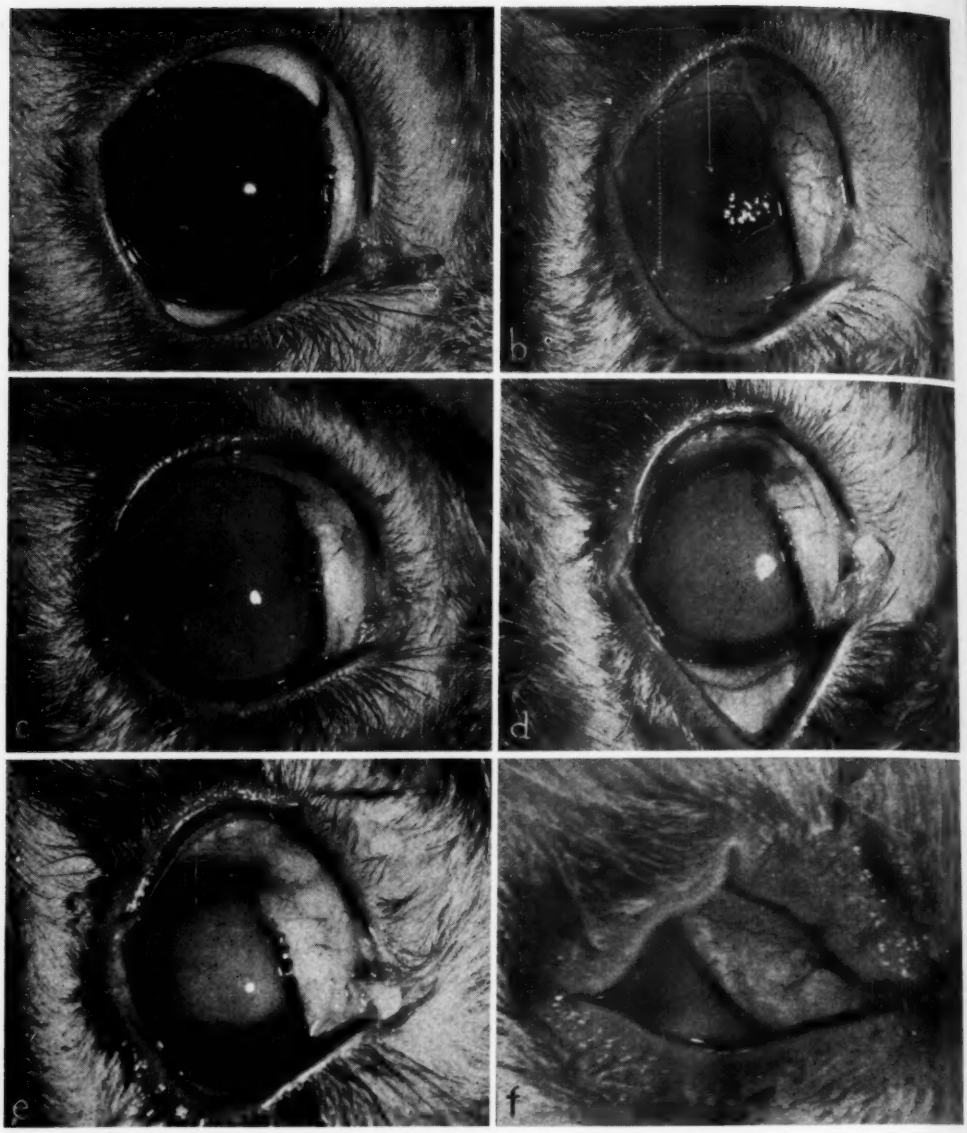

Fig. 4 (Guerry). Corneal changes after coagulation of both arteries. a, the eye before operation; b, 24 hours postoperative; c, 4 days postoperative; d, 9 days postoperative, beginning vascularization; e, 18 days postoperative, peak of vascularization; f, 1 year postoperative, phthisis.

vessels was effected by the diathermy. In those eyes wherein slight gross clouding of the corneas occurred it seems logical to presume that still more tissue damage resulted from the operation but not enough to give rise to actual destruction; hence the increase in Tyndall effect, number of cells in the aqueous, and the more marked hypotony. When marked opacification of the corneas occurred the tension was reduced to extremely low levels because of complete or relatively complete destruction of the ciliary processes, the dissolution of which gave rise to the tremendous amount of pigment seen free in the aqueous and deposited over the

surface of the endothelium. In these eyes the corneal thickening came about probably as a result of corneal imbibition of aqueous following severe damage done the endothelium by the quantitative and qualitative alteration of the aqueous. The diminution of corneal sensitivity in the eyes wherein marked opacification occurred may be attributed to the great corneal swelling that must result in undue pressure on the nerves.

Complete opacification of the corneas and phthisis bulbi resulted in most of the cases in which Wagenmann cut both long posterior ciliary arteries and many of the short ciliary vessels. The technique he employed was similar to that used when only a single artery was severed. He also described the vascularization of the cornea, which he likened to interstitial keratitis. In several of his cases corneal ulcers developed, panophthalmitis, and finally complete destruction of the globe. The fundus changes that he observed before corneal clouding obscured the picture, were much more pronounced than those seen after coagulation of both vessels. Shortly following operation, he found the retina had become whitened, the process beginning anteriorly and progressing rapidly until the entire retina had become involved. Some hours later, the retina became folded, giving rise to a condition which he described as resembling "brain gyri." The process could not be followed beyond this stage because of the marked corneal clouding.

In Wagenmann's animals these changes which far surpass any seen following coagulation of both long ciliary vessels, can only be attributed to the cutting, not only of those vessels, but also of a great portion of the short ciliary vessels, the latter of which supply all of the choroid posterior to the equator. The blood supply of the entire uveal tract was thus reduced to an insignificant trickle which was insufficient to prevent phthisis.

Microscopic pathology

In order to study the pathologic changes microscopically, the following procedures were carried out:

(A) A single long posterior ciliary artery was coagulated in each eye of eight rabbits by the same technique. At intervals of 24 hours, 72 hours, 2 weeks, and 2½ months, two animals were killed by a blow on the head, the eyes enucleated, fixed in Zenker's solution, and embedded in celloidin. Two eyes were then sectioned serially in the horizontal meridian and the other two in the coronal meridian through the treated areas.

(B) Bilateral coagulation of both long posterior ciliary arteries in eight rabbits was also carried out and the same method followed as with the preceding eight animals. The sections were stained by van Gieson's or modified Mallory technique.

Results. Twenty-four hours after coagulation of a single artery, the changes found microscopically were minimal. Slight dropsical swelling of the endothelium was present and there was a faint trace of fibrinous material in the anterior chamber. The iris on the side which had been operated on showed slight congestion, particularly of the greater arterial circle. The ciliary processes of the treated side were moderately congested and the ciliary endothelium appeared slightly indistinct, the changes resembling those seen in early cloudy swelling. In the treated area the retina and choroid were destroyed, and for a considerable distance anterior and posterior to this area the choroidal vessels were markedly congested. A few polymorphonuclear leukocytes were found in the retina and choroid adjacent to the treated area. The retina anterior to the treatment area

showed disintegration of the neuroepithelial layer, and some disturbance of the pigment layer, as shown by poor staining of the nuclei. After four days the dropsical swelling of the endothelium had disappeared. A small amount of fibrinous material could be seen in the anterior chamber and scattered over the endothelium. The iris was slightly more congested on the treated side whereas the ciliary processes were much more congested and the ciliary epithelium much more indistinct than at the 24-hour stage. In the treatment area the retina and choroid were destroyed and the sclera showed necrotic changes. The choroidal vessels were moderately dilated anterior and posterior to the treatment area, while anteriorly the retina revealed the same disintegration of the layer of rods and cones. The limbal vessels on the treated side were slightly congested as compared with those of the untreated side. Between the ciliary processes in one globe a small amount of fibrinous material was present. After two weeks there was only a faint trace of fibrinous material in the anterior chamber, and the endothelium was intact. The ciliary processes on the treated side showed slight congestion whereas the changes noted in the retina anterior to the treatment areas were less pronounced. Two-and-one-half months after operation the anterior chamber still contained a small amount of fibrin. There was slight depigmentation of the pigment epithelium of the iris and ciliary processes on the treated side, and the retina was found to show the same changes as at earlier intervals in the area anterior to the treatment zone.

In the series of animals wherein both arteries were coagulated, eight eyes were found to show marked opacification of the cornea, one eye moderate clouding, and seven eyes no clouding 24 hours after operation. The pathologic changes observed were found to vary in intensity, the most marked changes being seen in those eyes having the intense corneal opacification, and minimal changes being noted in the eyes devoid of gross corneal changes.

After 24 hours in an eye which had shown gross corneal opacification, the corneal epithelium was found to show an irregularity in thickness, the peripheral portions being reduced to a thin layer only two cells thick. Slight dropsical swelling was present in the endothelial cells, the nuclei of which stained poorly. The anterior chamber was shallow and almost filled with fibrinous material, cellular debris, and pigment clumps. The posterior surface of the iris had been completely denuded of its pigment epithelium, which lay roundabout in scattered clusters or as free granules. The ciliary processes were necrotic and naked, the epithelium and pigment layer having undergone complete dissolution. On one side between the ora serrata and the treatment area, the retinal architecture had been completely destroyed whereas on the opposite side, the neuroepithelial layer alone had become necrotic. The choroidal vessels were dilated both behind and in front of the treatment area. The changes noted in the treated area were similar to those where only one artery had been coagulated and consisted of complete destruction of the retina and choroid. The sclera in that region was also necrotic. Posterior to the treated areas the retina was not impaired.

In another eye which had shown moderate corneal opacification, the changes were less marked. Complete destruction of the ciliary processes was limited to one side and on that same side the retina between the ora serrata and treatment area was necrotic. Some destruction of the ciliary and pigment epithelium of the ciliary processes was seen on the

opposite side, but this was not pronounced. The retina on that side was intact. A moderate amount of fibrinous material was present in the anterior chamber, and the corneal endothelium showed slight dropsical swelling and indistinct nuclear staining.

Seventy-two hours after operation changes similar to those noted in the 24-hour stage were present but more advanced. The corneal endothelium was completely absent in places, and in other regions reduced to a reddish-staining granular layer (Mallory stain). A few remaining cells showed dropsical swelling. The cornea had become twice as thick as normal and the lamellae had become pale and swollen. The anterior chamber was shallow and filled with fibrin, while the iris had lost its pigment epithelium entirely. Only ghostlike traces of the ciliary processes remained. Pigment clumps were scattered through the aqueous and the region once occupied by the ciliary processes. Between the ora and treatment area the retina had undergone complete dissolution, but posteriorly it appeared normal.

An eye, the cornea of which remained grossly clear, after 72 hours showed changes comparable to those occurring after coagulation of only one artery. In this instance, pathologic changes were limited to slight dropsical changes of the corneal endothelium, the presence of a trace of fibrin in the anterior chamber, slight dispersion of the pigment of ciliary processes, and destruction of the neuroepithelial layer of the retina on one side between the ora and treatment area.

After two weeks the cornea was still twice as thick as normal and by now vessels could be seen throughout the anterior half of the stroma. The endothelium had disappeared completely and pigment flecks could be seen deposited directly on the naked Descemet's membrane. The anterior chamber was filled with fibrin. The iris and ciliary processes had been reduced to pigmented fibrotic stumps (fig. 5). The lens was completely cataractous, and scattered throughout were Morgagnian droplets. The vitreous

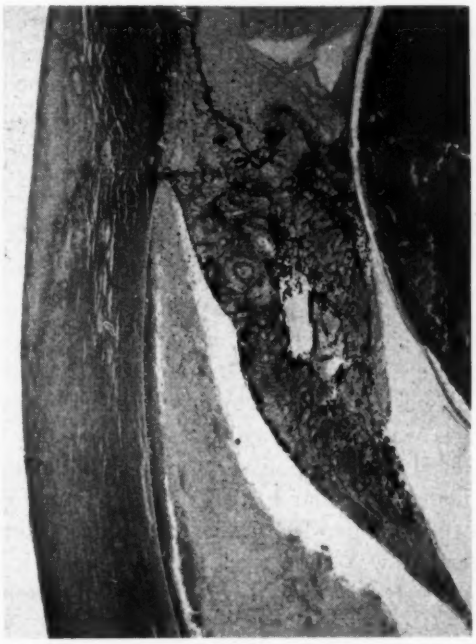

Fig. 5 (Guerry). Section showing a portion of cornea, iris, ciliary processes, and lens two weeks after coagulation of both arteries. The cornea is markedly thickened, the anterior chamber contains much fibrin, the iris and ciliary processes are reduced to fibrotic masses with pigment scattered about, and the lens is cataractous.

cavity was filled with fine, pink-stained, wavy material (Mallory's stain) resembling skeins of fine silk. The retina was completely destroyed anteriorly, but in the posterior part, although the neuroepithelium had undergone necrosis, the remaining retina retained its architectural characteristics fairly well. The pigment epithelium of the retina was destroyed anteriorly.

In one eye at this stage the changes paralleled those noted in the other eyes,

the corneas of which had remained grossly clear.

Two-and-one-half months following operation the globe had become shrunken to about two thirds normal size. The corneal endothelium was markedly vacuolated and covered by a homogeneous red-staining layer (Mallory's stain). The cells contained pigment which might have been phagocytosed. The cornea had regained its normal thickness, and the vessels had become much reduced in size. The anterior chamber was almost completely obliterated by anterior synechiae and what little space remained was filled with fibrin. Posterior synechiae were present also, and the pupillary area was completely occluded. Iris and ciliary processes were reduced to pigmented fibrotic masses, and the anterior surface of the iris was covered with Descemet-like material. The vitreous cavity was filled with material similar to that noted at the two-weeks stage. The anterior portion of the retina was destroyed, but the posterior part showed only neuroepithelial damage.

Discussion. The pathologic changes occurring after the coagulation of a single long posterior ciliary artery are minimal. For the most part they are transitory and the eye as a whole suffers but little. The damage done the retina, both in the treated area and between that area and the ora serrata, and the destruction of the choroid in the region where diathermy was carried out, are the only lesions which fail to undergo a completely reversible reaction, and these persist as definite lesions.

Wagenmann found pathologic changes similar to those described above. He found that the endothelium of the cornea showed slight granular degeneration and poorly staining nuclei. The anterior chamber contained fibrin in moderate quantities and the iris on the affected side was congested. He also described changes occurring in the neuroepithelial layer of the retina on the affected side as "loosening of the rods and cones." In some cases he found the vitreous to contain a small amount of fibrin. His studies following injection of gelatin into the carotid artery revealed that the iris and ciliary body on the side on which the artery had been cut were free from the injection mass whereas those structures were completely filled on the side that had not been operated on.

Following coagulation of both long ciliary vessels the changes noted in the different eyes vary tremendously. In half of the eyes complete destruction of the ciliary processes, necrosis of the iris, destruction of much of the retina, marked thickening of the cornea with vascularization, and finally shrinkage of the globe result. In an occasional case damage little more extensive than that noted when only one vessel was coagulated, occurred. In a little less than half of the eyes, the changes, though still marked, were not so extensive nor of such severity as to lead to phthisis. In these eyes only some of the ciliary processes became necrotic and only a small portion of retina was destroyed, these changes for the most part being limited to the structures of one side. In these cases collateral blood supply must be adequate or else one of the vessels must have been incompletely coagulated. Careful study of the treatment areas failed to reveal any patent lumen, but it must be understood that it is most difficult to determine what constitutes functional obliteration of a vessel seen in section.

The changes Wagenmann found after cutting both long posterior ciliary vessels, along with a large number of the short ciliary arteries, were similar to those seen following coagulation of both long ciliary arteries, although more pronounced. Extreme corneal thickening

with vascularization, destruction of the iris and ciliary processes, massive fibrinous exudation into both the aqueous and vitreous cavities, and complete destruction of the retina were usually found. In some instances where these changes were not so marked and where the globe did not become completely phthisical, he found the anterior ciliary vessels increased in size and apparently newly formed vessels arising from the conjunctival vessels at the limbus. In his opinion these collaterals prevented complete phthisis even though marked tissue destruction did take place. Following injection of gelatin into the carotid, the iris and ciliary processes were found to contain none of the injection mass.

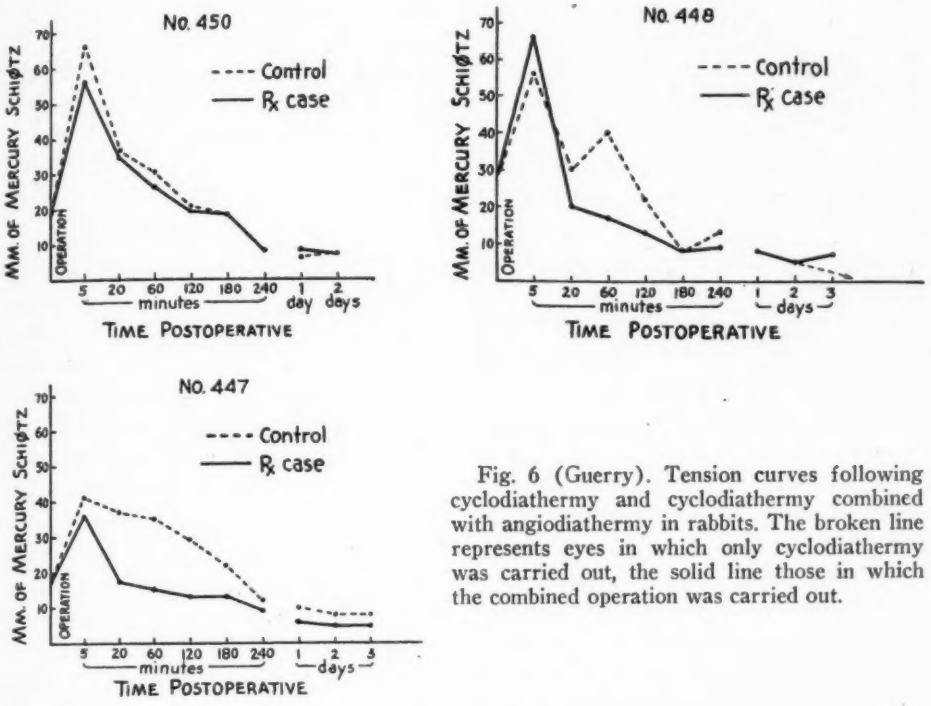

Fig. 6 (Guerry). Tension curves following cyclodiathermy and cyclodiathermy combined with angiodiathermy in rabbits. The broken line represents eyes in which only cyclodiathermy was carried out, the solid line those in which the combined operation was carried out.

Angiodiathermy and Cyclodiathermy

To determine the effect of combined angiodiathermy and cyclodiathermy the following procedure was carried out.

Method. A single eye in each of three rabbits was treated by cyclodiathermy and immediately thereafter a single long posterior ciliary artery was coagulated. The three fellow eyes were treated only by cyclodiathermy and served as controls. Bipolar diathermy current was employed in this experiment. After shaving the animal a neutral electrode was applied. The tension was then taken and the conjunctiva incised 5 mm. from the limbus, the incision extending around the upper half of the globe. The conjunctiva was dissected down to the limbus and the sclera cleaned over this area. Nonperforating cyclodiathermy was carried out, the Walker diathermy unit being set at 35. A flat Weve electrode measuring 2 mm. in diameter was used, four applications of six seconds' duration being made, two to either side of the superior rectus muscle. The treated areas appeared dull gray following the diathermy. Immediately after cyclodiathermy a long posterior ciliary artery was identified and coagulated, the Larkin modification of the Lacarrère electrode being employed. The conjunctiva was then replaced and the tension taken at intervals. In the control eyes cyclodiathermy was carried out, as with the fellow eye, but the long posterior ciliary arteries were allowed to remain intact.

Results. In one animal (no. 450) the

tension of both eyes rose rapidly to strikingly high levels and then fell over a period of two hours to normal, the fall then continuing for another hour until one half the preoperative level was attained. Similar abrupt tension rises were noted in the eyes of animals no. 447 and no. 448, but in these animals the tension of the eyes in which the combined operation had been carried out fell much more rapidly to a hypotonic level. The tension curves for these animals are shown graphically in figure 6. In all cases the tension remained low for about two weeks and then began to rise slowly until the preoperative level was reached on the seventeenth day to the twenty-first day.

Discussion. Weekers and Weekers[9] have noted the postoperative rise and fall in nonperforating cyclodiathermy carried out in normal rabbit eyes. The sudden rise is explained by them as being due to an immediate rapid dilatation of the uveal vessels. The fall, they contend, is due to an increase in absorption of the aqueous which has undergone considerable change in character. The explanation for the tension rise appears quite logical, but the explanation for the fall is highly unlikely. The more plausible explanation would seem rather to be a decrease in aqueous output due to the severe damage suffered by the ciliary processes.

In one animal (no. 450) the control and treated eyes followed a similar tension pattern but in two animals (no. 447 and no. 448) the tension fell much more rapidly in the eyes in which the combined operation had been carried out. This may mean that either the initial uveal dilatation was somewhat modified or else aqueous production was slowed down more rapidly than would be the case following cyclodiathermy alone. If angiodiathermy had been carried out prior to cyclodiathermy this effect might have been more striking.

The fact that tension curves of control and treated eyes followed a remarkedly similar pattern after the first few hours indicates that any effect angiodiathermy might have on the course of cyclodiathermy in these animals is transitory and limited to the first few hours. The return of tension to normal after three weeks would seem to indicate that either the ciliary processes are not destroyed in the treated region or else the remaining processes compensate by producing more aqueous.

Angiodiathermy in Man

After carrying out the experiments previously described on rabbits, the effect of angiodiathermy of a long posterior ciliary vessel was studied in man. In two cases of absolute glaucoma an attempt was made to carry out coagulation of the temporal long posterior ciliary artery after first detaching the external rectus muscle. No attempt was made to locate the artery, but diathermy punctures were blindly staggered in the region of the equator over the supposed course of the vessel. In both cases the tension was not affected in the least. I then found, after observing several eyes at the time of enucleation, that the temporal vessel is usually obscured by the attachment of the inferior oblique muscle, whereas the nasal vessel is more prominent and can easily be identified, once the internal rectus muscle has been detached.

In view of these findings a new procedure was followed. Two-percent procaine hydrochloride was injected retrobulbarly and in the region of the internal rectus muscle. The conjunctival sac was flushed with 25-percent argyrol, 1:1,000 adrenalin solution, and 4-percent cocaine. The speculum was inserted, and

the conjunctiva incised over the region of the internal rectus muscle. This muscle was then mobilized on a strabismus hook, a double-armed silk suture passed through its tendon, and the tendon cut free from the globe. A traction suture was passed through the tendon stump and the globe widely abducted. An Arruga detachment speculum was inserted, the sclera cleaned thoroughly, and the artery identified. Coagulation was carried out posterior to the equator with the Larkin modification of the Lacarrère electrode. The electrode tip was set at 1 mm., and the coagulation carried out by making perforations over the visible course of the vessel. The setting on the Walker diathermy machine was 45. Identification of the long posterior ciliary artery proved to be the most difficult part of the procedure. If the eye was well abducted, however, and care taken to clean the sclera thoroughly so that it became glistening white, identification was easily accomplished. In an occasional case, at the time of enucleation, the actual entrance of the vessel into the sclera was observed. The technique employed is shown in an artist's drawing, figure 7.

Case reports

Case 1. P. T., an Italian man, aged 75 years, was admitted to the Eye Institute, November 30, 1943, with the complaint of severe pain in the right eye of two weeks' duration. One year before, the patient had noticed that the vision in the right eye had become poor, and occasional episodes of pain and redness had occurred. He consulted his family physician, and "drops" were prescribed which "made the eye worse." Light perception had not been present for at least six months. The patient was seen in the Vanderbilt Clinic, the diagnosis of absolute glaucoma in the right eye made, and enucleation advised.

At the time of admission the patient's vision was: right eye no light perception, and left eye 20/70, unimproved with lenses. The tension was: right eye 65 mm. Hg, and left eye, 19 mm. The left eye was normal except for incipient cortical lens changes. The bulbar and palpebral conjunctiva of the right eye showed moderate injection. The cornea was very edematous and precluded detailed observation of other structures, but the pupil could be faintly seen fixed in mid-dilatation. There was no defect noted following transillumination. Following angiodiathermy, the cornea cleared. The iris was then found to be atrophic, bound down with posterior synechiae, and covered with newly formed

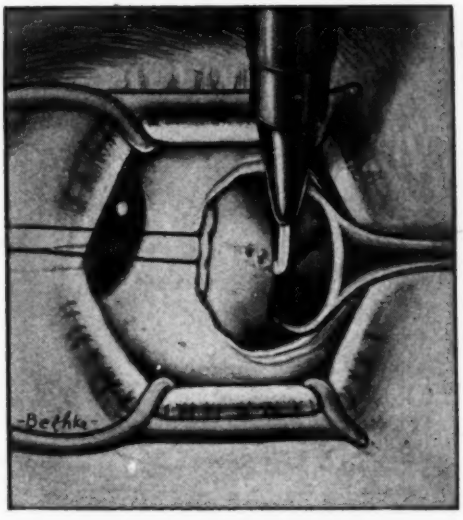

Fig. 7 (Guerry). Angiodiathermy in man.

vessels. A few old keratitic precipitates were seen on the endothelial surface. The anterior chamber was very shallow but no cells nor flare was present. The lens was cataractous, and no fundus reflex could be obtained. Although the patient was admitted for enucleation of the right eye, angiodiathermy was carried out to study the effect on the intraocular pressure.

At operation on December 1, 1943, no difficulty was encountered in finding the nasal long posterior ciliary artery. It was coagulated through the sclera well behind the equator. Figure 8 illustrates the tension curve following operation. From this it is evident that the tension reached a low of 31 mm. Hg on the third day only to be followed by a moderate rise for several days and then another drop to the 31-mm. level. The cornea cleared on the second postoperative day and remained clear until the eighteenth day, when it was again found to be edematous. The eye was comfortable throughout the entire postoperative course even though the tension reached preoperative levels. Corneal sensitivity was markedly impaired before the operation and no change was noticed

postoperatively. The patient was discharged on the nineteenth day, and though the tension remained high and the cornea cloudy, he was comfortable until February 1, 1943, when pain became troublesome. The eye was enucleated February 2, 1944, and pathologic studies are to be carried out.

Case 2. C. B., a colored man, aged 68 years, was admitted to the Eye Institute on December 27, 1943, with the diagnosis of absolute glaucoma of the right eye.

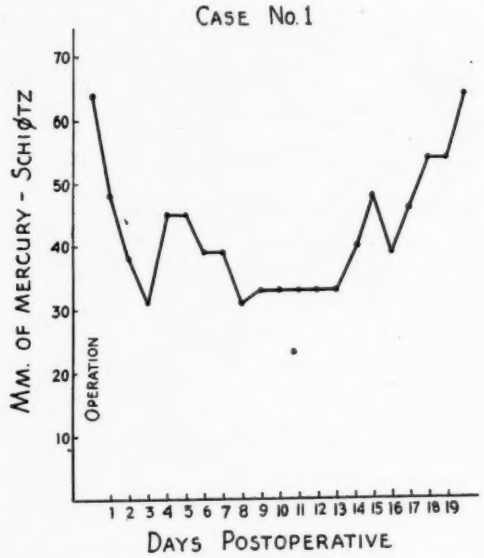

Fig. 8 (Guerry). Tension curve following angiodiathermy of nasal long posterior ciliary artery.

In February, 1942, an intracapsular extraction, left eye, was carried out following which the patient obtained 20/30 vision with correction. An extracapsular extraction was performed on the right eye in July, 1942. After operation the eye became irritable, and a dense secondary membrane developed. Glaucoma then set in, and in spite of treatment all light perception was lost. The tension remained high but for the most part the patient was comfortable. Angiodiathermy of a long posterior ciliary vessel was suggested as a possible means of lowering the intraocular pressure.

At the time of admission the patient was found to have vision of 20/30 in the left eye with correction, and no light perception in the right eye. Except for aphakia with complete iridectomy, the left eye was normal. The tension in the right eye was 100 mm. Hg and the cornea was extremely edematous. Through the corneal haze, the pupillary area was seen to be occluded by a dense whitish membrane, and the anterior chamber was completely absent below and shallow above. No fundus reflex could be seen. Transillumination revealed no defect. On December 28, 1943, angiodiathermy of the nasal vessel was carried out, the vessel being coagulated well behind the equator. The tension the next day had fallen from 100 mm. Hg to 17 mm., and the cornea had cleared completely. The upper and lower lids showed some edema, which was found to be due to sensitivity to sulfathiazole ointment employed at the time of operation. The postoperative tension curve is shown in figure 9. From this curve it is evident that the tension remained low for nine days and then gradually rose over a period of 10 days to the preoperative level. Throughout this time, and until January 28, 1944, the patient was comfortable. Pain then developed and the eye was enucleated on February 1, 1944. Pathologic studies are to be carried out. During the time the tension remained low the cornea became clear and remained so until the tension began to climb. The reaction fol-

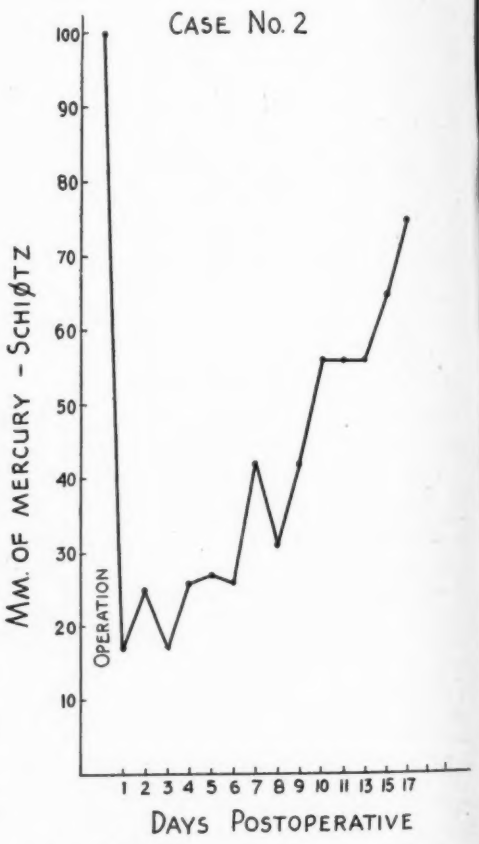

Fig. 9 (Guerry). Tension curve following angiodiathermy of nasal long posterior ciliary artery.

lowing the use of sulfathiazole ointment disappeared after four days.

Case 3. L. R., a 48-year-old Jewish man, was admitted to the Eye Institute September 3, 1943, for treatment of bilateral secondary glaucoma. His vision had been poor in the left eye for 15 years following a blow in that eye from a bowstring. Vision in the right eye had become impaired during the past two or three years during which time he was found to be suffering from moderately severe diabetes. His diabetes was controlled by 40 units of protamine zinc insulinate daily.

When first seen in the Vanderbilt Clinic on March 2, 1943, he was found to have vision of 20/70 in the right eye and 4/200 in the left eye, advanced diabetic retinopathy in both eyes, and subcapsular axial lens changes in the right eye. Although a guarded prognosis was given the patient asked that the lens of the right eye be extracted. An extracapsular extraction was performed on May 7, 1943, some two months after an uneventful preliminary iridectomy. The patient's postoperative course was smooth, but with correction, vision of only 20/200 was obtained in the right eye. In early July secondary glaucoma set in. This failed to respond to either miotics or mydriatics, and on July 16th cyclodialysis was carried out over the upper temporal quandrant. The postoperative course was uneventful, but the tension remained high. In August the tension was found to be elevated in both eyes, and in spite of intensive treatment with miotics remained uncontrolled. Acute glaucoma then developed in the left eye on September 3d, necessitating admission for treatment.

At the time of this admission the patient's vision was light perception in the right eye and 7/200 in the left eye. The tension was 48 mm. Hg in the right eye and 65 mm. in the left eye. Examination of the right eye revealed moderate corneal edema, an operative coloboma at the 12-o'clock position, rubeosis iridis, and a good fundus reflex but no detail. The cornea of the left eye was found to be very edematous, the pupil irregular and fixed in mid-dilation. Fundus detail was somewhat obscured but marked diabetic changes—such as, innumerable deep and superficial hemorrhages, edema of the retina, papilledema, areas of hard and soft exudate, and marked phlebosclerosis —could be seen. Intense miotic therapy was instituted without improvement, and on September 10th, perforating cyclodiathermy (Vogt type) was carried out over the lower one third of the left globe, some 20 perforations being made. Following this the tension rose considerably and the patient complained of severe pain, controlled by frequent hypodermic injections of morphine sulfate. Since the tension remained elevated in both eyes, cyclodiathermy was advised. Nonperforating cyclodiathermy of both eyes was carried out on October 1, 1943. The nasal long posterior ciliary artery of the right eye was coagulated at the same time.

Applications (nonperforating) with the Weve electrode were made over the ciliary body of the right eye, from the 1:30- to the 5-o'clock position. After the internus had been detached, the long posterior ciliary artery was identified and thoroughly coagulated just anterior to its entrance into the sclera. The strength of the current used for cyclodiathermy was that advocated by Albaugh and Dunphy;[10] that is, setting the Walker diathermy machine at 35, with each application eight seconds in duration. Applications were then carried out from the 11- to the 2-o'clock position on the left eye. The resulting tension curves are shown in figure 10.

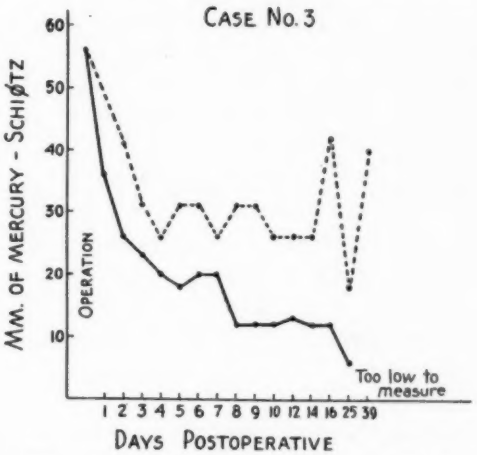

Fig. 10 (Guerry). Tension curves following angiodiathermy and combined angiodiathermy and cyclodiathermy. The broken line represents the eye in which cyclodiathermy alone was carried out, the solid line, the eye in which the combined operation was employed.

Discussion. In the two human cases of absolute glaucoma in which the nasal long posterior ciliary vessel was coagulated, a significant drop in tension occurred. The drop in case 1 reached its lowest level on the third postoperative day only to rise somewhat for two days. This was then followed by a fall on the fifth postoperative day, the tension then remaining low until the fourteenth postoperative day when it began to rise.

The preoperative level was reached on the nineteenth day after operation. The tension curve is not unlike that seen in the experimental animal following the same operation. The marked drop in tension in case 2 over a period of 24 hours to about one-fifth the preoperative level is striking. The gradual rise thereafter to preoperative level also parallels that seen in the experimental animals. No reason can be given for the more pronounced tension fall seen in case 2. In neither of these cases could the fundus be seen. Therefore, no fundus changes could be observed. Corneal sensitivity was markedly impaired before operation in both cases and no change was noted postoperatively.

Many cases following nonperforating cyclodiathermy, and particularly those of hemorrhagic glaucoma, develop an increased tension with intense pain postoperatively, and the tension falls to a low level only over a period of weeks. It was felt that in these cases, and perhaps in all cases of cyclodiathermy, this tendency to an initial tension rise might be controlled by angiodiathermy carried out at the same time.

In case 3, bilateral hemorrhagic glaucoma was present in a diabetic, and penetrating cyclodiathermy had been carried out over the lower one third of the left globe three weeks before. This had given rise to an increase in tension with severe pain. Cyclodiathermy of nonperforating type was then carried out in both eyes over an area of equal size. In the right eye the nasal long posterior ciliary artery was coagulated after cyclodiathermy had been carried out over the nasal region. From the tension curves it is evident that the tension fall following the combined operation was much more rapid and marked than in the eye wherein cyclodiathermy alone was employed. At the present time tension in the left eye has again become elevated, while that of the right eye is still too low to measure. The right eye has remained comfortable whereas the left is painful at times. Only light perception remains in either eye. Obscuration of the fundi by vitreous opacities prevented observation of the changes taking place. In this case also, corneal sensitivity was definitely impaired both before and after operation.

It may be suggested that the tension fall noted in these cases is not due to actual coagulation of the long posterior ciliary artery but instead to the perforation caused by the electrode at the time of operation. This argument can easily be disposed of by citing the two cases in which blind perforations caused absolutely no change in the tension. Further evidence to refute this is adduced by the fact that where posterior sclerotomy is performed with a resulting vitreous fistula, tension is rarely lowered for a period of over four or five days.

Conclusions

From these studies it seems evident that a transitory reduction of tension can be brought about in the human eye and in the rabbit eye by coagulation of a single long posterior ciliary artery. This is an innocuous procedure in the rabbit, and apparently in the human, although the human cases are too few in number to allow any definite conclusions to be drawn.

Angiodiathermy of a single long posterior ciliary artery should be of value in reducing the intraocular pressure in such cases wherein other operative procedures are contemplated and the pressure cannot be brought to a safe preoperative level. In other words, it should be a safer procedure than posterior scle-

rotomy, since the globe is decompressed more slowly and the infection hazard is much less.

When employed in conjunction with cyclodiathermy, angiodiathermy should prevent the initial tension rise that often proves so distressing.

These studies give added support to the teaching that the horizontal meridian should be carefully avoided during diathermy operation for retinal detachment. In case a single long posterior ciliary artery is inadvertently coagulated, hypotony results during the very interval when a normal or slightly elevated tension is desirable. If the experiments on rabbits can be applied to man, coagulation of both long posterior arteries might result in phthisis.

Summary

1. The results of coagulating the long posterior ciliary arteries of rabbits, singly and together, are compared with those obtained by Wagenmann after cutting these vessels.

2. Coagulation of a single long posterior ciliary artery was found to reduce the intraocular pressure in both the rabbit and human for a period of about two weeks. The procedure is relatively harmless.

3. Coagulation of both long posterior ciliary arteries in the rabbit resulted in phthisis bulbi in 50 percent of the cases.

4. The procedure is suggested as a rational substitute for posterior sclerotomy, and as a method of preventing the initial tension rise seen so frequently after cyclodiathermy.

5. The technique of coagulating by diathermy the nasal long posterior ciliary artery in the human is described.

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References

[1] Wagenmann, A. Experimentelle Untersuchungen über den Einfluss der Circulation in den Netzhaut- und Aderhautgefässen auf die Ernährung des Auges, insbesondere der Retina, und über die Folgen der Sehnervendurchschneidung. v. Graefes Arch. f. Ophth., 1890, v. 36, pt. 4, pp. 1-63.

[2] Fuchs, E. Beiträge zur normalen Anatomie des Augapfels. v. Graefes Arch. f. Ophth., 1884, v. 30, Abt. 4, pp. 1-60.

[3] Leber, Th. Die Circulations- und Ernährungsverhältnisse des Auges. Graefe-Saemisch Handbuch der gesamten Augenheilkunde. Ed. 2, 1903, p. 286.

[4] Maggiore, L. Struttura, comportamento e significato del canale di Schlemm nell'occhio umano, in condizioni normali e patologiche. Ann. Ottal., 1917, v. 45, pp. 317-462.

[5] Fortin, E. P. Investigations sur le glaucome. (Essais). Buenos Aires, A. Frascoli, 1939.

[6] Davis, F. A. The anatomy and histology of the eye and orbit of the rabbit. Trans. Amer. Ophth. Soc., 1929, v. 27, p. 401.

[7] Krause. Anatomie des Kaninchens. Leipzig, 1884.

[8] Baurmann, M. Ueber das Ciliarfortsatzgefäss system. Deut. Ophth. Gesell., Berlin, 1930, v. 48, pp. 364-371.

[9] Weekers, L., and Weekers, R. Mode de l'action des operations antiglaucomateuses, specialement de la diathermisation du corps ciliare. Ophthalmologica, 1942, v. 104, pp. 1-14.

[10] Albaugh, C. H., and Dunphy, E. B. Cyclodiathermy—an operation for the treatment of glaucoma. Arch. of Ophth., 1942, v. 27, pp. 543-557.

THE PRODUCTION OF LENS SENSITIVITY IN RABBITS BY BRUCELLA INFECTION*

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In an earlier report[1] it was shown that rabbits could be sensitized to lens substance by the potentiating effect of staphylococcal toxin. This sensitization was shown by cutaneous reactions to solutions of beef, pig, human, and rabbit lens; also by marked anaphylactic intraocular reactions when lens substance was liberated by needling the lenses of these sensitized animals. In applying these observations to an understanding of lens sensitization in humans the suggestion was made that a low-grade infection introduced into the lens at the time of extracapsular extraction and the coincident absorption of bacterial products and lens produced the sensitivity. This hypothesis was not subject to confirmation in the rabbit because of the varying intraocular reactions to staphylococci.

More recently, in studying the ocular pathology produced by brucella,[2] the idea suggested itself that this organism might be used to test the soundness of the aforementioned hypothesis. A strain of brucella, isolated from a mare with periodic ophthalmia, was used throughout these studies. The organism was first introduced into the anterior chamber of the eyes of rabbits. It produced varying amounts of intraocular inflammation, ranging from a destructive panophthalmitis to mild chronic iritis that eventually cleared up with few or no sequelae. Later, inoculations were done by lightly scratching the cornea with an infected 25-G. needle. At the end of 48 hours all eyes appeared normal. After that time and up to seven days later about 75 percent of the animals developed iritis of varying intensity and clinically resembling periodic ophthalmia in horses. It is interesting that this observation challenges the strongly held opinion that organisms cannot pass the relatively unbroken cornea without inducing destructive abscess formation. These results are cited briefly to orient the reader as to the pathologic possibilities of brucella.

The following experiments were made to test the validity of this hypothesis. Six rabbits were injected in the right eye by the following method: A 25-G. needle attached to a syringe was first dipped into a 48-hour broth culture of brucella. This needle was then introduced into the anterior chamber, and the aqueous, under its own pressure, was allowed to flow into the syringe. The needle was then inserted into the lens and the syringe emptied. This technique produced marked lens damage, rupturing the lens and expelling considerable lens substance. Within 24 hours there was marked intraocular inflammation in all eyes, which continued for about three weeks, when all the eyes were quiescent. Each week after the initial inoculation, the animals were tested for lens sensitivity by the cutaneous injection of bovine lens solution, 1:100. There was no real evidence of cutaneous sensitivity. Occasionally a small nodule could be felt on the following day, but these were definitely doubtful reactions. All of the rabbits developed positive cutaneous reactions to brucellin, a substance analogous to Deny's bouillon filtrate. Three of the rabbits developed precipitin titers for lens extract as high as 1:40.

After four weeks the lenses of the left eyes were needled as has been described,

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except that the needle was sterile. Four of the rabbits developed violent anaphylactic intraocular reactions within 24 hours. Sections from eyes removed at various intervals showed histologic pictures similar to those seen in the eyes sensitized by the action of staphylococcal toxin. There was no suggestion of brucella infection in these eyes. Parenthetically, it is admitted that this infection does not produce a particularly characteristic histologic picture. Six normal rabbits were needled in parallel with the aforementioned animals and showed only the usual traumatic cataracts.

This experiment was repeated on 12 rabbits with the same essential technique and results, except that the needlings were done eight weeks after the original inoculations and the rabbits were observed at varying intervals up to one year. The same violent inflammatory reactions were observed within 24 hours after needling the normal left eye, and at the end of one year all of the left eyes showed evidence of the previous trauma in well-defined cataracts, posterior synechiae, and scarred irises.

At irregular intervals preceding and following the needlings of the left eye, blood cultures for brucella were made, with entirely negative results. This was done to answer the obvious criticism that organisms, circulating in the blood, localized in the damaged lens.

If the results of the blood cultures rule out this chance, these experiments suggest that lens sensitization can be produced by infecting a damaged lens. They do not suggest that brucella is the offending organism, although, from the recent findings of the role of this organism in human ocular disease, they may occasionally play the sensitizing role.

References

[1] Burky, E. L. Experimental endophthalmitis phaco-anaphylactica in rabbits. Arch. of Ophth., 1934, v. 12, p. 536.

[2] ——. The role of brucella in ocular disease. Amer. Jour. Ophth., 1939, v. 22, p. 1210.

OPHTHALMOSCOPIC CLASSIFICATION OF HYPERTENSIVE DISEASES*

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Richard Bright,[4] over 100 years ago, described hypertension associated with kidney disease, and his concept of hypertension existed until 1896, when Clifford Albutt[1,2] described hypertension as being due to some vasoconstricting mechanism which led to arteriolar sclerosis and was not primarily associated with kidney disease. Janeway,[12] in 1913, stated he believed that the disease was one of the small blood vessels and that the kidney disease was a secondary manifestation. This belief was generally accepted until 1932, when Goldblatt,[9] began various experiments on dogs and monkeys and found that, by constricting the renal artery on one side, he could produce hypertension resembling that occurring in man. He showed that the causative factor was ischemia of the kidney, the degree of hypertension depending upon the degree of occlusion of the renal artery. He demonstrated further that malignant hypertension could be produced by constriction of both renal arteries. Here again the degree of hypertension was dependent upon the degree of constriction, up to nearly total occlusion, for when total occlusion occurred, or when both kidneys were removed, no hypertension developed.

Tigerstad and Bergmann,[18] in 1899, discovered that extracts from the kidney contained a protein that would produce a rise in arterial pressure. In 1939 Page[14] purified rennin and showed that alone it would not cause hypertension, but that there existed in the blood a rennin activator which, when combined with rennin, produced angiotonin. Angiotonin caused the vasoconstriction and was, therefore, responsible for the hypertension. The work of Page has demonstrated that there is formed in the blood an inhibitor substance which may be a valuable therapeutic aid in hypertension.

Since 1932 much work has been done on the hypertensive states, and considerably more is known concerning pathogenesis. Nevertheless no definite conclusions can be drawn from this knowledge, for there are many factors that may produce the same type of hypertensive disease.

Fishberg's conception is not conclusive, but seems to define the situation at present. "The concept of essential hypertension includes those cases of chronic hypertension which neither clinically nor anatomically can be demonstrated to have evolved from an antecedent inflammatory disease of the kidney or of urinary obstruction"—so that we have adopted the term "essential benign hypertension," but no definite classification has been accepted. The classification by Weiss[23] defines the various known groups, which he outlines as follows:

I. Hypertension of organic vascular origin with renal ischemia:
   1. Congenital malformations

* Read at the seventy-ninth annual meeting of the American Ophthalmological Society, at Hot Springs, Virginia, June, 1943.

a. Coarctation of the aorta
b. Hypoplastic renal artery
c. Polycystic kidneys

2. Inflammatory vascular diseases
a. Glomerulonephritis
b. Pyelonephritis
c. Periarteritis nodosa
d. Arteritis—"rheumatic"
e. Disseminated lupus erythematosus

3. Degenerative vascular diseases
a. Arteriosclerotic occlusive lesions of the renal artery ("essential hypertension")
b. Renal arteriosclerosis ("essential hypertension")

II. Hypertension of organic vascular origin without renal ischemia: Diffuse arterial and arteriolar sclerosis

III. Hypertension of nonorganic vascular origin:

1. Endocrine disorders
a. Pituitary dysfunction and neoplasms (Cushing syndrome)
b. Adrenal dysfunction and neoplasms (cortical and medullary)
c. Certain types of toxemias of pregnancy

2. Nervous disorders
a. Cerebral trauma
b. Cerebral neoplasms
c. Poliomyelitis
d. Rare vascular diseases of the brain

This classification does not include malignant hypertension, which, we believe, should be in a separate classification, as it is, in our opinion, a disease entity.

The ophthalmic literature contains many articles concerning the ocular findings in the various hypertensive states, but here also the nomenclature and classification of diseases are very confusing. There would seem to be utter confusion in the minds of the ophthalmologists as to which of the various terms to use, for in no section of this country is there a common language. There are certain terms that should be in universal usage, and we should attempt to adopt some common classification.

The ophthalmoscope affords a better knowledge of the hypertensive states than can be obtained from any other single examination, and in most cases it makes the diagnosis and prognosis possible. Surely no internist would be without the aid of such an examination, and he does expect from the ophthalmologist a clinical interpretation of these findings.

In many hypertensive states the first ophthalmoscopic finding is that of angiospasm. This group we classify as "angiospastic hypertension." These spasms may occur in early benign hypertension, malignant hypertension, or in toxemia of pregnancy. Mylius,[15] Wagener,[20] and Hallum[21] have described the various types of spasms seen in toxemia of pregnancy, and there can be no doubt that the spasm of the arterioles is responsible for the hypertension and that the degree of spasm is indicative of the degree of hypertension. Just what the mechanism in the development of the arteriolar spasm is remains unexplained. In spite of the experimental work of Goldblatt to the contrary, we feel that there must be some central vasomotor influence. This type of hypertension usually comes on very suddenly, and the blood pressure, as in the organic group, gives a high systolic and diastolic reading. This high diastolic pressure is an indication of the amount of peripheral resistance or, in other words, the degree of arteriolar spasm, since most observers agree that constriction of the arterioles is responsible for the hypertension. These angiospastic states have become generally recognized, and certain transitory cerebral lesions have been described by Pal[17] as cerebral vascular crises and by Kauffman[13] as angiospastic in-

sults. Pain and paresthesias of the extremities are probably angiospastic in origin, as is Raynaud's disease and erythromelalgia, as described by Westphal and Bar.[24] There can be no doubt that these contractures of the arterioles in the retina are angiospastic. A very striking example of such spasms not heretofore described is seen in the arterioles that supply an area of acute exudative chorioretinitis, in optic neuritis, and also, at times, in choked disc. These spasms may lead to organic changes in the vessel wall, and Wagener states that such organic changes may begin within 10 days, though we believe that a vessel may be angiospastic for months and then apparently return to normal in ophthalmoscopic appearance.

Many cases of essential hypertension begin as angiospastic states and gradually develop organic changes and so become arteriosclerotic. Patients who develop one-sided kidney disease are likely to be in this angiospastic group, and many of the cases of malignant hypertension, if seen early, would show simply a marked angiospasm. Toxemia of pregnancy represents the most classic picture.

In angiospastic states, hemorrhages, exudates, and edema do not occur. However, when the spasm becomes complete, edema does appear. So, in the angiospastic patients who develop exudates and hemorrhages, we believe that the latter are definite evidence of considerable kidney damage.

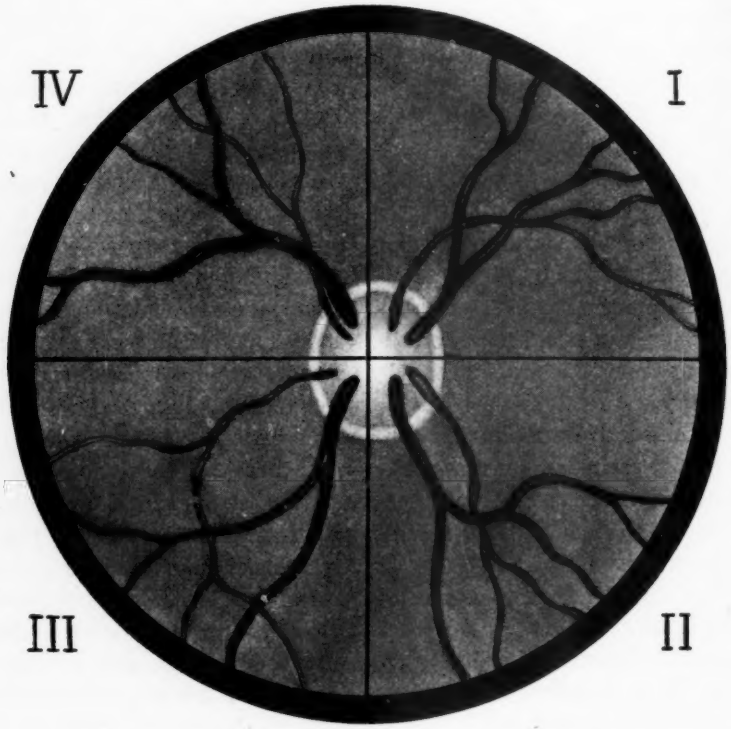

Fig. 1 (Clay and Baird). Arteriolar sclerosis.

The spasms of the arterioles vary, depending upon the degree of hypertension, so that a grading of the degree of spasm is most important. Therefore, as an aid to clinical interpretation, we divide angiospastic spasms into four grades:

Grade I. There is only a slight irregular contraction seen in one or two arterioles.

Grade II. The contraction is found in most of the vessels and there is some irregularity in caliber.

Grade III. There is marked reduction in caliber and usually there are marked irregularities. The higher the tension, the more irregularities there are, or the greater are the spasms.

Grade IV. The arterioles are in a state of complete spasm—mere threads, almost obliterated, as seen in complete spasms of the central artery.

If this classification is used, when one states that he has a case of angiospastic hypertension Grade II, one knows immediately the degree of involvement as well as the degree of hypertension.

In the angiospastic group, edema, hemorrhages, and exudates do not occur, as a rule, unless the spasms are in Grade III or IV. These changes are due to severe angiospastic damage in the kidney, and the patient is approaching malignant hypertension. The term to be used is "angiospastic hypertensive neuroretinopathy." The term "retinitis" should not be used, as the process is not inflammatory.

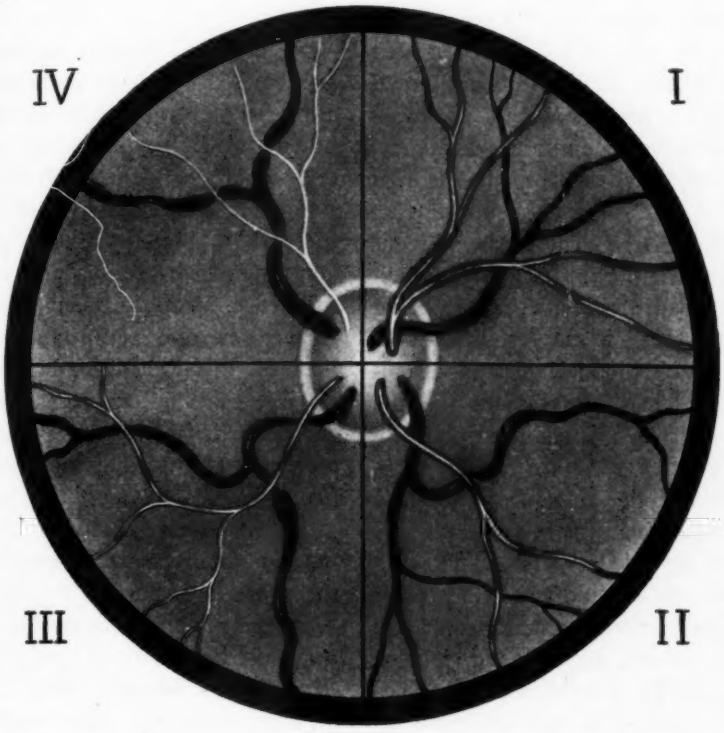

FIG. 2 (Clay and Baird). Arteriolar sclerosis.

Spasm can easily be differentiated from sclerosis. In spasm there is little loss of transparency, the reflex stripe is not exaggerated, and there is no arteriovenous compression. It is impossible to tell when a spastic vessel begins to take on organic changes, and not until some compression of the vein is evident can one state that arteriolar sclerosis is present.

Eighty percent of all hypertensive pa-

tients should be classified as manifesting "essential benign hypertension." Following the classification of Weiss, as previously outlined, this group occurs largely under the heading of *Degenerative vascular diseases.* Of all the other groups, the one condition next in importance in the production of hypertension is glomerulonephritis. Essential hypertension may occur at any age from 8 to 80 years, but the large majority of cases occurs between the ages of 40 and 50. There is no known cause for the hypertension in this group. There is a strong hereditary tendency, however, and renal ischemia is the real factor.

This hypertensive group has in common a high systolic and usually a high diastolic pressure, the average being 160/100. However, it may read 300/180. A higher average blood pressure is usually encountered in the malignant and the angiospastic groups. The blood pressure generally fluctuates, there being some cases in which the pressure will vary from 30 to 50 mm. in a few hours. This variation is found in persons showing marked evidence of emotional instability. Patients with marked fluctuation in pressure always exhibit angiospastic changes in the fundus. The majority of these patients die of cardiac or cerebral involvement, only about 20 percent of deaths being due to renal diseases. As has been stated, the results of the examination of the kidneys is essentially negative, the urine is normal, the blood chemistry is normal, and kidney clearance tests are negative. These patients are usually free from symptoms—slight headache may be the only symptom for years. For that reason they are most frequently seen first by the ophthalmologist, who finds arteriolar angiospasm the only early positive finding. Bell and Clawson[8] and many others believe that spasms are responsible for the beginning of the disease: following spasm, the vessels become sclerotic. Fishberg has shown that in practically every case the arterioles are the vessels involved; the arteries rarely show much involvement until very late in the hypertensive disease. The arteriolar disease may show fairly uniform distribution throughout the body, but the kidney will exhibit more marked disease of the arterioles.

In essential benign hypertension the arterioles are always involved, either by angiospastic or by sclerotic changes. No other fundal lesion is present except occasionally a few isolated retinal hemorrhages. These are the result of a sudden increase in blood pressure and are not accompanied by edema or exudates. We, therefore, strongly believe that the term "arteriosclerotic retinitis" should never be used. In Grade II or III of the sclerosis one may find a complete or partial central-vein thrombosis. This ophthalmoscopic picture does not indicate a bad prognosis.

The arteriolar sclerosis is the important factor in essential hypertension, and the degree of sclerosis represents the exact stage of hypertensive disease, so that it is most important to grade the sclerosis.

Arteriolar disease is seen in most cases of hypertension. The early sclerotic changes are often difficult to see with the ophthalmoscope, but as the disease progresses they become easy to recognize. We classify the vessels seen in the fundus as arterioles. Therefore, the term "arteriolar sclerosis" is used entirely. We believe that the marked reduction in caliber and the irregularities are manifestations of previous spasms, and we call this condition "arteriolar sclerosis, angiospastic type." The arteriolar disease should be divided into four stages, representing the degree of sclerosis:

Grade I. There is loss of translucency; the light reflex is increased.

Grade II. The light reflex is very distinct, and there is a slight arteriovenous compression.

Grade III. The caliber is reduced, and the reflex stripe is extremely conspicuous. There is marked arteriovenous compression, with distal dilatation. Grades II and III represent the copper-wire arterioles.

Grade IV. This is the silver-wire arteriole; the vessel has become practically obliterated and is a mere thread.

Where there is a marked silver-wire condition of the vessel, there has usually also been extreme spasm. Fishberg stated that endarteritis obliterans is most frequently seen in chronic glomerulonephritis, and Volhard and Fahr[19] believe that the endarteritis is the result of severe angiospasm.

To grade arteriolar disease in this way is simple and it gives the internist the type of information he needs. For example, when it is stated that the patient has an arteriolar sclerosis Grade III, angiospastic type, the internist will know at once that the patient has had a fluctuating and rapidly developing type of hypertension; that he has reached the latter stage of his disease, and probably will die of coronary disease.

Arteriolar sclerosis or angiospasms, or a combination of both, are always associated with essential benign or malignant hypertension, although a patient may have recovered from an acute hypertensive episode and his fundus fail to show any evidence of sclerosis. It is possible, on the other hand, for slight residual changes to remain in the vessels of the fundus and the patient may not present any elevation of blood pressure, but such a patient is potentially a hypertensive case.

Senile arteriolar sclerosis exists, and is not accompanied by any marked degree of hypertension. In fact, there may be only a slight elevation of the systolic pressure, and the individual will show arteriosclerosis. The arterioles are usually reduced in caliber, but the caliber is very regular, the vessels are much straighter, and the reflex stripe is not so conspicuous as that seen in patients of the essential hypertensive group. In this senile arteriolar sclerotic group patients are much more likely to show sclerosis of the choroidal vessels. Senile arteriolar sclerosis is not just an aging process, for on ophthalmoscopic examination many persons past 70 years of age will show entirely normal arterioles.

The majority of the patients suffering from essential hypertension seen by us in the hospital develop one of two things: cardiac failure or renal disease. The group with cardiac failure will not show any lesions of the fundus other than the advancing arteriolar disease, whereas in the group with the developing kidney disease, showing a slight increase in albumin and nonprotein nitrogen, the retinas will show flame-shaped hemorrhages, a few exudates, and occasionally slight edema of the nerve head and retina. In our opinion these changes are always definite evidence of severe kidney damage, although it is true that occasionally, as was shown by Keith and Wagener,[14] the results of the kidney examination may be negative, and yet in almost every one of these cases before death there will be definite evidence of kidney disease. This picture is entirely different from that of malignant hypertension, and should not be confused with it by calling it the malignant phase of essential hypertension. The term that we believe is best descriptive of this group is "secondary nephritic hypertensive neuroretinopathy."

Malignant hypertension is more frequently seen in younger individuals and somewhat resembles the more severe cases of toxemia of pregnancy. These patients' blood pressure is much higher, and, usually within two to four years,

they die of a kidney disease.

Fahr,[7, 8] Wilson and Byrom,[25] and Ellis[5] have demonstrated that this group presents a different pathologic picture—a much more severe disease of the arterioles—which they describe as an arteriolar necrosis with greater changes in the kidney. Goldblatt's[10] experiments showed that such a hypertension could be produced by compression of both renal arteries, and the degree of hypertension would increase with the degree of compression. There are a few cases of essential hypertension, chronic glomerulonephritis, and angiospastic hypertension in which a malignant hypertension develops, but the majority of these cases are a distinct entity. The fundal picture of malignant hypertension is diagnostic. There is more marked edema of the nerve head and retina, and hemorrhages and exuduates are more extensive. The edema is so marked that it is often difficult to see the vessels, but the arterioles show marked angiospasms with a less severe degree of sclerosis until late in the disease. The grading of this group by Wagener[21] and by Wagener and Keith[22] is best followed.

The term we believe most suited for this group is "malignant hypertensive neuroretinopathy."

The development of increased intracranial pressure in hypertension is still unexplained, but Cushing[6] demonstrated that the arterial pressure rose progressively only when the tension within the calvarium approached the level of the diastolic pressure. Such patients develop a choked disc, and usually this is seen in the malignant group; associated with it are all the other changes previously described.

Occasionally one encounters an interesting phenomenon in the essential benign group—a choked disc without any other findings than the severe degree of arteriolar sclerosis of the angiospastic type.

Nephritis will not produce changes of the fundus to any great extent, with the occasional exception of slight fluffy exudates and mild retinal edema, but when hypertension develops in such a case, more especially chronic glomerulonephritis, characteristic changes will appear. These cases do not show the severe edema of the nerve head and retina, and the exudates are more prominent, being of the waxy, glistening type. Fewer hemorrhages are present, and the arteriolar disease is not so marked. The term that best describes this group is "primary nephritic hypertensive neuroretinopathy."

The classification here outlined seems to be one that would include simply all the hypertensive states, briefly as follows:

I. Angiospastic hypertension
   1. Arteriolar angiospasm, Grades I to IV
   2. Angiospastic hypertensive neuroretinopathy

II. Essential benign hypertension
   1. Arteriolar sclerosis, Grades I to IV
   2. Arteriolar sclerosis, angiospastic type
   3. Secondary nephritic hypertensive neuroretinopathy

III. Malignant hypertension
   1. Malignant hypertensive neuroretinopathy

IV. Nephritis with hypertension
   1. Primary nephritic hypertensive neuroretinopathy

A short paper that attempts to cover the entire subject of hypertension must needs be sketchy. We hope to cover each subject classification more fully in subsequent papers.

Our studies have convinced us that the information gained from the ophthalmoscopic examination is of greater diagnostic value than that of any other examina-

tion; that frequently with this information alone an accurate diagnosis can be made; and that the classification advocated in this paper is simple and readily mastered.

There is so much confusion in the minds of the average ophthalmologist, and there are so many different classifications, that we feel that the greatest need now is for a commission to be appointed by the three national societies to formulate proper nomenclature and classification of the hypertensive diseases.

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References

[1] Albutt, Sir Clifford. Disease of the arteries, including angina pectoris. London, Macmillan & Company, 1915, v. 1, p. 534.
[2] ———. Jour. Hunterian Soc., 1896, v. 38.
[3] Bell, E. T., and Clawson, B. J. Arch. Path., 1928, v. 5, p. 939.
[4] Bright, Richard. Guy's Hosp. Rep., 1836, v. 1, p. 388.
[5] Ellis, H. Lancet, 1938, v. 1, p. 977.
[6] Cushing, H. Bull. Johns Hopkins Hosp., 1901, v. 12, p. 290.
[7] Fahr, T. Deutsches Arch. f. klin. Med., 1920, v. 134, p. 366.
[8] ———. Centralbl. f. allg. Path. u. path. Anat., 1916, v. 27, p. 481.
[9] Goldblatt, H., and Hahn, J. R. Experimental observation on the malignant phase of essential hypertension. In Blood, Heart and Circulation. The Science Press, 1940.
[10] Goldblatt, H. Ann. Int. Med., 1937, v. 11, p. 69.
[11] Hallum, A. V. Jour. Amer. Med. Assoc., 1927, v. 20, p. 180.
[12] Janeway, T. C. Amer. Jour. Med. Sci., 1913, v. 145, p. 625.
[13] Kauffman. Hypertension. Leipzig, 1926, p. 64.
[14] Keith, N. M., Wagener, H. P., and Kernohan, J. B. Arch. Int. Med., 1928, v. 41, p. 141.
[15] Mylius, Karl. Funktionelle Veränderungen am Gefässsystem der Netzhaut. Berlin, S. Karger, 1928, p. 82.
[16] Page, I. H. Newer aspects of experimental hypertension. In Blood, Heart and Circulation. The Science Press, 1940.
[17] Pal, J. Gafässkrisen. Leipzig, S. Hirzel, 1905.
[18] Tigerstad, R., and Bergmann, P. G. Arch. f. Physiol., 1898, v. 8, p. 223.
[19] Volhard, F., and Fahr, T. Die Brightsche Nierenkrankheit; Klinik, Pathologie und Atlas. Berlin, J. Springer, 1914.
[20] Wagener, H. P. Jour. Amer. Med. Assoc., 1933, v. 101, p. 1380.
[21] ———. Trans. Amer. Ophth. Soc., 1927, v. 25, p. 349.
[22] Wagener, H. P., and Keith, N. M. Arch. Int. Med., 1924, v. 34, p. 374.
[23] Weiss, Soma. The clinical physiologic characteristics of arterial hypertension. In Blood, Heart and Circulation. The Science Press, 1940.
[24] Westphal and Bar. Deutsches Arch. f. klin. Med., 1926, v. 151, p. 1.
[25] Wilson, C., and Byrom, F. B. Lancet, 1939, v. 1, p. 136.

Discussion

Dr. P. J. Leinfelder (Iowa City): Dr. Clay's paper has been of unusual interest to me for at least two reasons, one being that he makes some statements with which I differ, and another, that he presents so much material with which I am in agreement. In the first place, there is no question about his statement that the literature with its terminology is confused. Our graduate students repeatedly come to us with the complaint that they cannot make head or tail out of hypertensive vascular disease. As a result we have had develop our own classification which, I am happy to say, is quite a bit in agreement with Dr. Clay's outline. We can all, I am sure, take exception to parts of this outline; yet, for practical purposes, it seems to me most clearly to express the state of affairs as we see them in the study of large numbers of hypertensive patients.

There is one point I would like to speak of which Dr. Clay did not mention, but to which, I feel certain, he pays considerable attention, and that is the status of the exceedingly small arterioles, the vessels of the third and fourth branches of the central artery. We find very frequently that the first changes are seen in those small vessels and consist of local irregularities and narrowings which may progress to such an extent the small vessels are seen with extreme difficulty. In the earliest stages the changes in hypertensive vascular disease seem to be confined exclusively to these very fine vessels. As the disease advances, the vascular changes progress down the vessels toward the nerve head and in the late stages involve the largest arterioles. It is impossible for anyone to say that at the present time we know all there is to know about hypertensive vascular disease. I feel that Dr. Clay's paper contains an extreme amount of practical information which deserves careful study, and that all of us can profit from his experiences and from his classification.

Dr. Theodore L. Terry (Boston): Dr. Clay's paper is timely and of considerable interest. My only question is whether his proposal goes far enough. The recent advances in surgery upon the sympathetic nervous system and upon discovery and removal of functioning adenomata of the adrenal glands, not only tend to control, but, in some instances, to cure the younger patient with severe hypertension. The surgeons, cardiologists, and internists most active in selecting those hypertensives who should be subjected to the surgery know of the importance of careful detailed eye examination as an adjunct to their examination and they are as concerned as we over the selection and us of proper terminology. I think they as well should be consulted before any definitive terminolgy is adopted by us, even though they already are guided to a great extent by the ophthalmologist's report on the fundus findings.

Dr. Jonas S. Friedenwald (Baltimore): I think that Dr. Clay has brought up a very important problem in emphasizing the difficulties in nomenclature in this field. Perhaps it may help us slightly toward the solution of this nomenclature problem if we understand a little bit more fully how these confusions have developed. There are two aspects that seem to me to have contributed very seriously to the development of confusion in our study and knowledge and classification of vascular diseases. In the first place, vascular disease has been classified primarily from two points of view: the clinical point of view, and the pathologic point of view, and these two classifications have never satisfactorily coincided. The pathologist recognizes at least two primary vascular diseases: (1) the intimal disease, which consists of proliferation of the endothelium with fatty plaques in the endothelium, which are first called arteriosclerosis, and later called atherosclerosis, and have still other new names from time to time; (2) the disease recognized by the pathologist as the hyaline degeneration of the media of the artery. Since this second form occurs most often in the smallest vessels it was originally called arteriolar sclerosis, and further names have been given to it from that point of view. Both of these conditions can, on occasion, affect vessels of the same size, so that classifying the two diseases on the basis of the size of the vessel attacked, and basing the retinal classification on the fact that the arterial vessels we see in the retina are all arterioles, do not in themselves resolve the confusion. The major vessels in the retina may at some time show in some cases atheromatous changes and be associated with what used to be called athero-

sclerosis in the larger vessels. They may in other cases show hyaline degeneration and be associated in the rest of the body with arteriolar sclerosis or hyaline degeneration of the media of the vessels in other organs. Thus the overlapping of these two diseases in the medium-sized vessels constitutes one of the barriers to a reasonable classification.

The second aspect of the classification difficulty is that while either of these disease processes may be primary, each one can indirectly cause the other disease process to develop. For instance, if a patient starts out with what used to be called arteriolar sclerosis—that is, hyaline degeneration of the media of the arterioles—he can get hyaline degeneration of an arteriole in the vasa vasorum of a coronary artery. The coronary artery suffers in nutrition as a result of this hyaline change of its nutrient arteriole, an atheromatous plaque may develop on the intima of that vessel and the patient may die from a cardiac infarction. Thus atherosclerosis can in many instances occur as secondary to the hyaline change of the small arterioles. Conversely, a patient may start out with what is primary atherosclerotic disease, have an atheromatous plaque in his renal artery, and get ischemia of the kidney and secondary malignant hypertension and arteriolar changes in the small vessels. It is the fact that these two diseases are so confused in their course, and that many patients when they reach the terminal stages have both diseases, one primary and the other secondary, one mutually fortifying the other, that makes the nomenclature so confusing.

Dr. John N. Evans (Brooklyn): It seems to me that our problem goes much further back than just to classifications. We have to begin, if we are going to make progress, with a better understanding of the basis on which we draw our inferences. If we look at the retinal vessels and say, as Dr. Clay did, "vasospasm," we cannot directly justify that diagnosis from anything we have seen. What we can say is, "These vessels look narrow." Then ask ourselves, "What are the many, many things which can make vessels look narrow?" In quizzing our medical students, we may say: "What are the distinguishing appearances of choked disc and papillitis?" It is easy to make a tabulation of over 100 factors and conditions which can make the nerve head look blurry. If we cannot decide what features differentiate one of these from the other, how are we going to make progress in classifying? And so if this classification is to be accepted it will have to begin with acceptable criteria from which to draw inferences.

Dr. Clay (closing): I appreciate this discussion. I want to emphasize this point: that we consider the benign hypertensive group by far the largest group of hypertensive disease, and in these 10 cases, the chances are, that had the fundi been examined shortly before death, we would only have seen an arteriolar sclerosis, probably Grade II. The large majority of individuals whom we see with hypertension show a gradual increase in the degree of arteriolar sclerosis, and that degree of sclerosis definitely represents a grade of hypertension, in our opinion. If there are irregularities in the caliber of the arterioles we believe that it is an indication of a previous spasm, and that the individual has had a fluctuating type of hypertension.

KERATOCONUS POSTICUS CIRCUMSCRIPTUS

A CASE REPORT

GEORGE WISE, LT. (J.G.), (MC), U.S.N.R.

Shortly after reading Leopold's[1] paper, the following case of keratoconus posticus circumscriptus was encountered. Because of its rarity (only four cases,[2] including Leopold's, have been previously reported in the English literature), its description was felt to be warranted.

C. E. P., a white sailor, 24 years of age, was admitted to the hospital on January 11, 1944, complaining of blurred vision of the right eye. He had first become aware of the reduced vision of the right eye at the time of his naval entrance examination four months previously, this being the first eye examination of his life. Prior to that time, he had worked as a farmer and had had no eye trouble. There was no history of trauma. The past medical history was negative as was the family history, except for tuberculosis on his father's side. The patient had never been in contact with a known, active case of tuberculosis. He felt that the vision might always have been poor in this eye and that he might never have become aware of it except for the naval examination.

Examination. O.D., the vision was 20/50. Aside from the corneal lesion, to be described, and a small patch of medullated nerve fibers below the right disc, this eye was entirely normal.

O.S., the vision was 20/20. This eye was entirely normal except for a mild congenital ptosis, the upper lid crossing the limbus at the 2:30- and 9:30-o'clock positions.

The pupils, muscle balance, and fields of both eyes were normal. The tension of each was 20 mm. Hg (McLean).

The right globe was white. There was no corneal staining with fluorescein, and the anterior chamber was clear. The anterior surface of each cornea had normal curvature and the cornea of the left eye was entirely negative.

Under direct illumination, the cornea of the right eye showed a large, eccentric, nebulous opacity occupying more than one-half the total corneal area (fig. 1). With the slitlamp, the posterior corneal surface in the nebulous area showed an irregularly scalloped curvature with a grayish rather than transparent appearance. The cornea, at its thinnest portion, was reduced to one-fourth its normal thickness, and this area was a little below the opacity's center. The stroma and the anterior cornea were quite normal, and there was no increase in the prominence of nerve fibers. It was readily seen that the corneal nebula was due to the changes of the posterior corneal surface. The lesion approached the limbus between the 7:30- and the 9:30-o'clock positions, but left a completely transparent zone between itself and the limbus, similar in all respects to the clear area seen in dystrophies. To be sure of this observation, the patient's head was turned to one side, and, with the light beam crossing the questionable area at right angles, the fact was demonstrated beyond any doubt.

Near the periphery, at the 8:30-o'clock position, there were a few Hassal-Henle bodies, but none was noted elsewhere. In very thin cross sections observed with the slitlamp, the edge of the lesion showed a brushlike change extending slightly into the stroma from the posterior surface (fig. 1). No corneal pigmentation was noted. Retinoscopy was unsatisfactory,

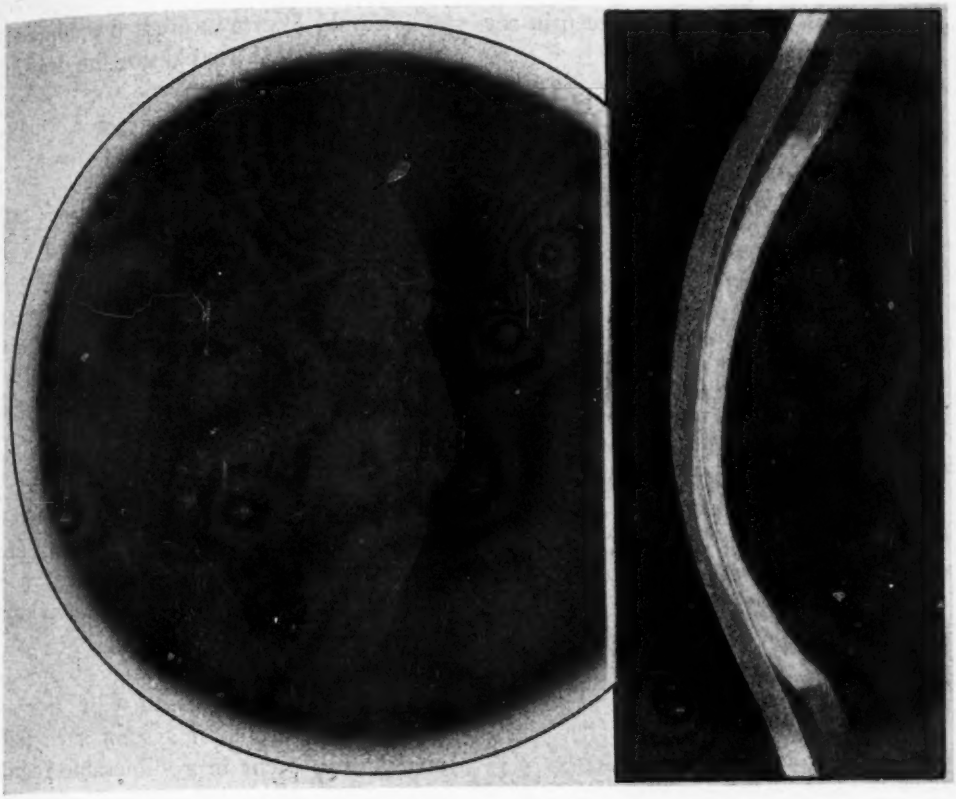

Fig. 1 (Wise). Keratoconus posticus circumscriptus.

for, with the pupil dilated, its shadow simulated that of a dislocated lens.

Vision under homatropine cycloplegia was: O.D. with a −0.50D. sph. ⊂⊃ +2.25D. cyl. ax. 150°, 20/30; O.S. with a −1.00D. sph. ⊂⊃ +0.75D. cyl. ax. 90°, 20/15.

The general physical examination revealed nothing of significance. Blood count, Kahn test and urine were normal.

Comment

This case appeared to be very similar to that of Dr. Leopold. Several factors seemed worth stressing: the eccentricity of the lesion, the scalloped irregularity of the posterior-surface curvature, the normal anterior cornea, and the fact that the opacity was due to changes of the posterior corneal surface. Neither this case, nor any of the others described, have been shown conclusively to be acquired. A traumatic etiology has not been clearly established in any case, nor has the progressive nature of the lesion been demonstrated with certainty in any. The appearance in the case herein reported suggested neither of these.

It is interesting that a cornea can have only one-fourth its normal thickness and still withstand the intraocular pressure and maintain its normal anterior curvature. This suggests that true keratoconus is due to some abnormality of the entire corneal structure.

One other interesting feature of this case is the way in which the lesion stopped short of the limbus, leaving a clear zone

similar in all respects to that seen in corneal dystrophies.

I should like to express my appreciation to Mr. E. G. Bethke for the drawing to illustrate this case.

Bibliography

[1] Leopold, I. H. Arch. of Ophth., 1943, v. 30, Dec., p. 732.
[2] (a) Stallard, H. B. In discussion of Butler, T. H. Trans. Ophth. Soc. United Kingdom, 1930, v. 50, p. 551.
(b) Butler, T. H. Brit. Jour. Ophth., 1932, v. 16, p. 31.
(c) Ingram, H. V. Trans. Ophth. Soc. United Kingdom, 1936, v. 56, p. 263.

CLINICAL STUDIES IN ANGIOSPASM*

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New York 21

The question as to whether angiospasm is brought about by neurogenic factors or through chemical mediation of toxic, humoral, or hormonal influences is today answered by most observers by the statement that both are at work either independently or together in any individual case.

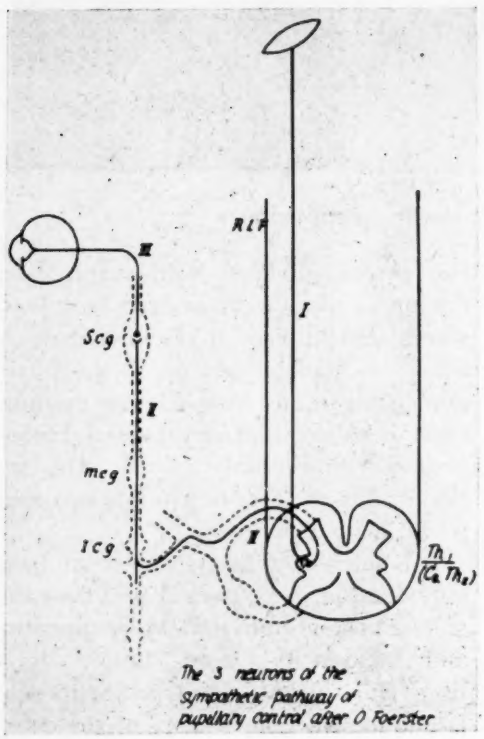

Fig. 1 (Givner). The three neurons of the sympathetic pathway of pupillary control (after O. Foerster).

In regard to localized angiospasm the possibility that in a person with an especially labile vasomotor system such phenomena can occur in a vulnerable region is not difficult to comprehend when we think of other localization phenomena such as occur in the Shwartzman reaction. On a basis of local sensitivity a small portion of a vessel can be picked out for angiospastic attack.

Although not proved it is most likely that all blood vessels are supplied by sympathetic fibers. The fibers that arise from the lateral ganglionic system are controlled by the spinal center for vasoconstriction (fig. 1). These, in turn, are under the control of the hypothalamus. A method of approach to disturbances in the region of the hypothalamus—namely, pupillography—was utilized in one of the cases of angiospasm to be reported and is recorded not for the conclusions in the findings but to call attention to this extra aid in the armamentarium of angiospastic research.

In patients with a vasolabile apparatus in whom the difference in temperature of

* Read before the New York Society for Clinical Ophthalmology, December 6, 1943.

the abdomen and extremities, taken in a draft-free room, exceeds 6 degrees, blocking of the tibial nerve with a 2-percent solution of procaine hydrochloride can cause a relaxation of the arterial wall together with an increase in the temperature of points distal to this block by at least 2 degrees and usually more. It does not seem unreasonable to expect some dilatation in the retinal arteries after retrobulbar injections of 2-percent novocaine in cases of acute angiospastic phenomena of nervous orgin if the patient is seen early enough and if endarteritic changes and organized blocking are not present. This paper presents three cases of angiospasm each of which is representative of a type.

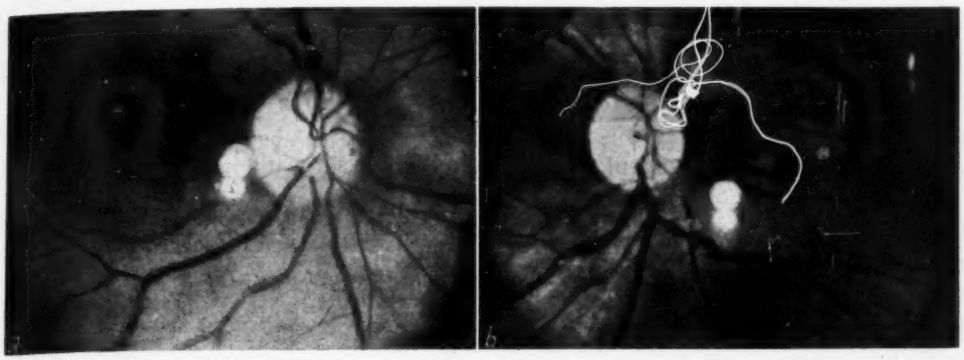

Fig. 2a (Givner). Fundus photograph showing the normal artery-vein (2:3) relationship.
Fig. 2b. Fundus in case 1, showing arterial angiospasm; artery-vein relationship 1:3.

Type 1

Evanescent retinal spasm producing visual disturbances in a patient with so-called "angioneurotic diathesis."

Case 1. The patient, C. S., a woman, aged 28 years, complained of flashes of light occurring for the past two years. Different parts of the visual field were affected. Since her seventeenth year the vision in the right eye had blurred momentarily and she would see only part of an object; then vision would return. Her past history revealed that when she was 13 years old she had been told that her blood pressure was 180/. At the time of this examination it was 142/100. One year ago she had had an angioneurotic edema of both lids after an emotional upset. Urinalysis revealed albumen, which disappeared after the lid condition subsided. The patient was seen during one of the attacks of flashes and blurred vision.

It should be remembered that the average width of retinal arteries at this patient's age should be inferonasal 70, superonasal 79, superotemporal 110, and inferotemporal 116 microns (fig. 2a). Figure 2b shows the narrowing of all the arteries as compared with the normal.

At the time of an attack 10 c.c. of blood was drawn from her vein, centrifuged, and the serum injected into the tail artery of a white rat. A few words on the details may not be amiss.

A 1-percent solution of sodium amytal is given intraperitoneally, 0.8 c.c. per 100 gm. In five minutes the rat is asleep. The pupils are easily dilated with 2-percent homatropine hydrobromide. The average blood pressure in a rat of 200 gm. is about 110/80. The normal blood volume of such a rat is about 12 c.c. Normally a 5-c.c. injection, by so markedly increasing the blood volume, may raise the pressure as

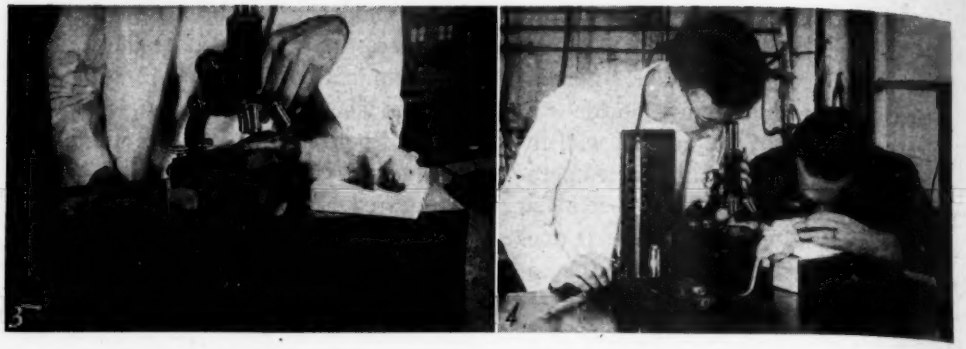

Fig. 3 (Givner). Method (of Duncan, Hyman, and Chambers) for studying blood pressure in a rat.

Fig. 4. Synchronous fundus study and blood pressure readings.

high as 20 points, but no vasoconstriction of the arterioles appears either in the retina or in the small vessels of the web. The method of examination is that devised by Duncan, Hyman, and Chambers[1] with a special condensing lens for a light source (figs. 3 and 4). After fixing the leg, the web arterioles can be observed with 100 magnification compared to the 14 magnification seen with the ophthalmoscope. All blood flow is stopped; then it begins to come back but in a jerky fashion. This is considered the systolic pressure. When the blood flow is continuous the diastolic pressure is noted.

One cubic centimeter of the patient's serum was injected. In two minutes the pressure had begun to rise, and the retinal arteries showed a narrowing. The fundus was easily observed with the ophthalmoscope, by means of a +12.00D. lens. In eight minutes a rise of 35 mm. was noted. The control pressure showed a rise of 5 mm. It is interesting to note that the retinal arteries show the change before the blood pressure is at its highest and are relaxed before the blood pressure returns to normal.

Pupillographic studies of this patient showed the following as reported by Dr. Otto Lowenstein: "The type of reaction to light, the type of aniscoria, and the presence of a good psychosensory dilatation while psychosensory restitution phenomena are poor indicate the presence of a central sympathetic lesion."

Type 2

Disseminated angiospastic retinopathy—a term which I have coined for this type, having in mind Horniker's suggested central angiospastic retinitis, which was improved to central angiospastic retinopathy by Gifford and Marquardt.[2]

Case 2. The patient, F. W., a plumber, aged 52 years, was first seen in September, 1940. He stated that the vision in his left eye had become blurred in the central portion although his peripheral vision was clear. He described his vision as clouds with many colors. There has been no improvement in the condition.

Examination disclosed spasm of the temporal vessels of the left eye with edema involving the macular region. Six weeks later the right eye became affected. There was slight blurring in the lower portion of the visual field in the right eye. Examination disclosed an angiospasm of that portion of the superotemporal artery nearest the disc with edema localized to the area surrounding this spasm. The left eye showed an angiospasm of one of the branches of the superotemporal vein surrounded by localized edema. In addition, there were macular edema and degenera-

tive changes, and an accompanying central scotoma. On November 1, 1940, new areas of localized spasm and edema were noted in the right eye. In the old area the vessel was now seemingly patent and the edema disappeared. The inferotemporal artery was involved in the angiospastic and edematous process. The left eye showed the originally spastic venule opened and a new branch involved. In short, these spastic and relaxing phases covered the fundi of both eyes in a disorganized fashion. The areas where the spasm had lasted too long were left with retinal degeneration until the final picture was loss of vision in both eyes (fig. 5). The patient's blood pressure was 130/72. There was no family history of hypertension nor allergy. The patient smoked about 20 cigarettes daily. He was tested with tobacco extract 1/100 to 1/200 c.c. intradermally with saline control. The result was negative as were all other tests for allergy. Because of his occupation lead was considered a possible etiologic factor but both urine and blood were normal—urine 0.003 mg. per 24 hours, blood 0.006 mg. per 100 c.c. Blood smears showed no stippling. Blood chemistry, including cevitamic-acid determination, phosphatase-N.P.N., urea nitrogen, calcium, and cholesterol was normal. All foci of infection were removed. The vascular status after a study of the nail bed was reported by Dr. A. Wilbur Duryee as normal. Venous pressure, right arm, was 58 mm.—normal; left arm 52 mm.—normal. The patient was treated with vasodilators, sodium nitrite, acetylcholine both intravenously and intraorbitally to no avail. The administration of bellergal, foreign proteins, intravenous injections of sodium chloride, and various vitamin preparations had no beneficial effect.

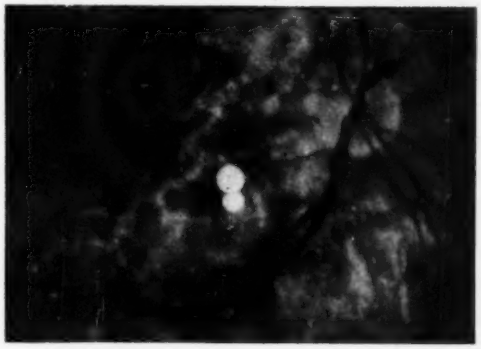

Fig. 5 (Givner). End result of retinal degeneration after diffuse angiospastic retinopathy.

TYPE 3

Angiospastic episodes of hypertensive retinopathy.

Case 3. M. B., a man, aged 53 years,

Fig. 6 (Givner). Papilledema in case 3 resembling malignant hypertension but only an angiospastic episode in hypertensive neuro-retinopathy.

Fig. 7. From the same patient 2 years later with all evidences of angiospasm gone—post-papilledemic atrophy of nerve head.

was seen on June 19, 1941, with the typical picture of malignant hypertension (fig. 6) but with normal blood chemistry. His blood pressure was 168/120. A report was sent that the prognosis as to life was bad and 6 to 12 months were given, as 79 percent die within that time. Two years later the patient came in with a complete absence of all angiospastic phenomena (fig. 7). His vision was 20/200 because of postpapilledemic atrophy, but the general status was fair. The blood pressure was 220/135. The blood chemistry was normal. The only treatment that may have had some effect was vigorous foreign protein (typhoid intravenously given by Dr. Harry Solomon). A personal communication from Dr. Henry P. Wagener noted, "I have seen several cases of the type you describe. I think there should be some way of differentiating these pictures when we see them. We are dealing with an acute angiospastic episode and not with true retinitis of malignant hypertension. So far as I can recall, the blood chemistry was normal in these cases." Such subsidence was noted as far back as 1859 by Liebreich.

It is interesting in this connection to recall the report of Smithwick and Castleman[3] on patients on whom they have operated (not necessarily involving cases of malignant hypertension) in eight of whom normal renal arterioles were found.

It would seem to me that just as the level of the hemoglobin helps to differentiate fundi of perplexing cases of chronic glomerular nephritis in the last stages from malignant hypertension (in that a low hemoglobin and low red blood cell content is the rule in the former and many times a high hemoglobin in the latter), so in like fashion, the blood chemistry should guide the ophthalmologist in giving a prognostic report. If the blood is normal, the condition is possibly only an angiospastic episode in hypertensive retinopathy; if abnormal, malignant hypertension may be considered to exist in view of the fundus picture.

In conclusion it is suggested that investigations on these cases in the future include if possible pupillographic studies to help gain more information as to the part the hypothalamus plays.

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References

[1] Duncan, Hyman, and Chambers. Jour. Lab. and Clin. Med., 1943, v. 28, April, pp. 886-889.
[2] Gifford, S. R., and Marquardt, G. Arch. of Ophth., 1939, v. 21, Feb., p. 211.
[3] Smithwick, R. H., and Castleman, B. Jour. Amer. Med. Assoc., 1943, v. 121, April 17, pp. 1256-1261.

THE FREQUENCY OF SQUINT*

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According to the literature and other available sources of information, the frequency of squint, is not a common subject. In the general population, including both children and adults, it is almost impossible to estimate it accurately, nor does it seem to be significant except from an academic standpoint. Of major importance to orthoptic technicians, however, is the incidence of different types of squint and the possibilities regarding their treatment.

The New York Society for the Prevention of Blindness has made many statistical reports on the incidence of strabismus among children in schools. In different cities in the United States the incidence of squint in children has been variously reported as beng 1.5 percent, 0.8 percent, and 1.5 percent.[1] Children under 2 years of age, from 2 to 7 years of age, and in the preschool age showed an incidence of squint of 1.3 percent, 2.4 percent, and 4.7 percent, respectively.[2] The British Board of Education, London, reported that of 1,199,291 school children examined in England and Wales, 1.3 percent had squint.[3]

The incidence of squint in a busy clinic is next called to our attention. During the month of July the Department of Ophthalmology of the Cleveland Clinic saw 1,804 patients. Of these patients 271 were new, and 89 of them were sent to the Orthoptic Division for muscle-balance examination. Nineteen or 7 percent of the new patients had squint, which is approximately the incidence to be anticipated in new cases. During a year the incidence may vary and may even be slightly less, since July is a vacation month when one would expect to see more muscle anomalies in children than at other times of the year.

The frequency of squint cases seen in the Orthoptic Division alone is much higher: 21 percent of the new patients had cases of squint.

It is interesting to note the relative incidence of squint in different age groups. As would be anticipated, the greatest incidence is in the younger age groups where the fusion faculty is very often faulty or not fully developed. In the Public Health Report of July 17, 1925, the number of children with squint in the United States according to age was estimated as follows:[4]

Age (years)	Number	Percent
Under 5	172,000	1.5
5 to 9	157,000	1.25
9 to 14	120,000	1
15 to 19	57,000	0.5

In an abstract of an article, "The incidence of squint at various ages," by T. Stewart Barrie,[5] the percentage of strabismus cases at different ages was estimated to be as follows:

Age (years)	Percent
5	2.01
7	2.18
10	1.79
13	1.37

Deductions made from these figures were that

1. There is a 9-percent increase in the number of cases of strabismus between the ages of 5 and 7 years.
2. There is a uniform decrease in the

*Read before the third annual meeting of the American Association of Orthoptic Technicians, Chicago, Illinois, October 10, 1943.

cases of strabismus from 7 to 13 years.

3. From the shape of the graph, the decrease is likely to continue for some years after 13 years of age.

4. Thirty-two percent of the cases of strabismus at 5 years of age showed no obvious strabismus at 13 years.

Even in a relatively small group of squint cases examined at the Cleveland Clinic during one month, the same decrease in squint was observed as the age increased.

This definite decrease in the frequency of squint as the age increases brings up the question of what happens to the squint of these children as they become older. If untreated, does the angle of squint lessen as certain anatomic features of the head and eye mature, such as a decrease in the amount of hypermetropia? Do some children develop fusion much more slowly than do others, or are so many children being treated for squint that the incidence decreases as the age increases? Children do not just "outgrow" squint, and early treatment will give the best results in developing single binocular vision.

Dr. Frank N. Knapp,[6] in an article entitled "The economic importance of squint in children and its effect in after years," made a survey in April and May of 1929 in a mining company; 4,424 men were examined. He found that 4.3 percent of men suffered from squint out of a total of 339 men referred for examination and refraction. Of these, 2.6 percent had divergent squint and 1.7 convergent. Ten percent of those examined had amblyopia ex anopsia. Since 4.3 percent had squint and 10 percent had amblyopia ex anopsia, apparently many of the men with low vision in one eye had straight eyes. If the eyes of any of these men had been straightened by surgical means, or remained straight because of normal balance in a fusion-free position, there evidently had been no orthoptic treatment to develop binocular vision, and the patient kept suppressing the amblyopic eye the rest of his life. This percentage of men with amblyopia ex anopsia shows a great economic loss and emphasizes the importance of orthoptic treatment at an early age.

In the foregoing survey it will be noted that the cases of divergent squint were much more common than the cases of convergent squint. This, of course, was in an adult group. The higher frequency of exophoria and convergence insufficiency, which we have noted at the Clinic and which is so common in adult cases, inadvertently strengthens the theory that divergence of the eyes in a position of rest is more the rule than so-called balance or orthophoria; and when blindness occurs in one eye, a latent divergence becomes manifest.

In Dr. Knapp's survey of public schools in Duluth, 288, or 2.35 percent of a group of 12,253 children, had squint.[6] Of these, 217 were found to have convergent squint. In another Public Health Report, by Selwyn D. Collins,[4] the percentage of strabismus in a group of 12,134 school children was 0.91. Of these 0.73 had internal strabismus as contrasted with only 0.17 percent with external strabismus.

Most writers are agreed that concomitant convergent squint occurs most frequently in children. Dr. Bielschowsky,[7] in his "Theory of heterophoria," states that in children up to 15 years of age a convergent strabismus develops after the loss of binocular vision five times as frequently as in persons of 30 years or older. According to Dr. Allen[8] of Iowa City, the presence of convergent strabismus in children may be explained by the fact that most children are hyperopic.

Of the 166 cases seen at the Cleveland Clinic which were analyzed for this re-

port, 115 cases, or 69 percent, were of convergent squint: 36 cases, or 22 percent, of divergent squint, and 15 cases, or 9 percent, of vertical squint. Of course, many of the cases of convergent and divergent squint were associated with hypertropia. The cases were classified according to the greatest amount of deviation.

The various kinds of squint were classified as monocular, alternating, accomodative, and paralytic. Monocular squint was most common both in the convergent and divergent cases, the convergent monocular being 67 percent and the divergent monocular being 69.5 percent. Next in frequency were the cases of alternating squint, 26 percent of which were convergent, and 19.5 percent divergent. Of the convergent series 4.4 percent were accommodative (third in frequency of convergent squints). Of the paralytic squints, 11 percent were cases of divergent squint and only 2.6 percent of convergent squint. There was a higher percentage of both abnormal retinal correspondence and amblyopia ex anopsia in patients with convergent squint as compared with those with divergent squint.

Some interesting points relative to the age incidence were that

1. Of the 115 cases of convergent squint which were analyzed, the onset in all but 2 cases occurred between birth and 7 years of age, the majority occurring at preschool age.

2. In 36 cases of divergent squint 8, or 22 percent, developed after 7 years of age.

This reveals that 1.7 percent of the cases of convergent squint developed after the age of 7 years as compared with 22 percent of the cases of divergent squint. The fact that 52, or nearly half, of the patients with a convergent squint which developed in childhood were adults at the time of examination indicates that the squint tends to persist in later years unless treatment is begun at an early age.

There is not a significant difference in the incidence of deviations of the right eye and left eye. Of the cases of convergent squint 41 had a right-eye deviation and 45 a left-eye deviation, and of the cases of divergent squint, 18 had a right-eye deviation and 10 a left-eye deviation. In a group of 62 hypertropias and hyperphorias, 23 had a right hyperphoria or tropia, 30 had a left hyperphoria or tropia, and 9 were bilateral. This does not seem to be of significance, although in a larger number of cases, there may be a greater difference.

Almost all of the patients with squint wore glasses. Only a few had anisometropia. In the cases of convergent squint only 7 percent were myopic, the rest being hyperopic. Thirty-one percent of the patients who had divergent squint were myopic, 69 percent being hyperopic. In July, 1925, Selwyn D. Collins[4] reported the incidence of strabismus and difference in visual acuity in the two eyes. He found that the incidence of squint increased as the visual acuity decreased in equal vision. In unequal visual acuity the greater the difference in the two eyes, the higher the incidence of strabismus. The incidence of strabismus in these cases of unequal visual acuity increased more than in those with equal visual acuity. Where vision was 20/30 or better in one eye and 20/70 or worse in the other, the incidence of strabismus was increased as much as 10.45 percent as compared with 2.52 percent in equal visual acuity of 20/70 or worse.

Another type of squint is caused by blindness or partial blindness of the eye from various causes, such as corneal opacities, traumatic or congenital cataract, lesions of the retina or choroid, and so forth. When the eye becomes blind and

the patient no longer has fusion to hold the eyes parallel, the eye deviates out or in, or remains straight according to the position of rest, or the fusion-free position. In children the eye usually turns in, and in adults it usually turns out. These pathologic cases are sent to the Orthoptic Division for examination only if the patient wishes to have the eyes straightened for the cosmetic effect.

The frequency of squint may be summarized as follows:

1. In children the incidence is usually between 1 and 2 percent.

2. Squint is more frequent in younger children than in older children or adults.

3. In school children convergent squint has a higher incidence. In adults divergent squint has a higher incidence.

4. The frequency of squint according to type is as follows:

a. In convergent squint.
   (1) monocular
   (2) alternating
   (3) accommodative
   (4) paralytic

b. In divergent squint
   (1) monocular
   (2) alternating
   (3) paralytic

Paralytic squint is more frequent in divergent than in convergent squint.

5. There is a higher percentage of abnormal retinal correspondence and amblyopia ex anopsia in convergent than in divergent squint.

6. According to the time of onset of squint 1.7 percent of the cases of convergent squint develop after 7 years of age as compared with 22 percent of the cases of divergent squint.

7. There is no significant difference in the deviating eye in convergent, divergent, or vertical cases.

8. The refractive error is hyperopic in most cases, although there are more patients with myopia in the cases of divergent squint than in those of convergent squint.

9. The incidence of strabismus increases as the difference in visual acuity becomes greater between the two eyes. Strabismus also increases as the visual acuity in both eyes decreases, but not so markedly.

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References

[1] Relation between physical and mental status of children. U. S. Pub. Health Rep., July 19, 1929.
Report for year ending June 30, 1926. The Board of Public Education, Division of Medical Inspection of Public Schools, Philadelphia, Pa.
Emerson, Wm. R. P. Physical defects in 1,000 children. Amer. Jour. Dis. Child., 1927, May.
[2] Rude, A. E. Physical status of preschool children, Gary, Indiana. U. S. Child. Bur., 1922, no. 111.
Vision of preschool children. National Society for the Prevention of Blindness, no. 66.
[3] 1929 Annual Report, Chief Medical Officer, British Board of Education, London.
[4] Collins, S. D. Strabismus and defective color sense among school children. Reprint No. 1031, Public Health Report, Washington, D.C., July 17, 1925.
[5] Barrie, T. S. Trans. Ophth. Soc. United Kingdom, 1922, v. 42, p. 273. (Data taken from examination findings of Medical Officer, Clackamannan Education Authority.)
[6] Knapp, F. N. Economic importance of squint in children and its effects in after years. Minnesota Med., 1931, v. 14, April, p. 324.
[7] Bielschowsky, A. Lectures on motor anomalies. Dartmouth College Publications, Hanover, New Hampshire, 1940.
[8] Allen, J. H. Squints and squint training. Pub. Health Nursing, 1936, v. 28, Feb., p. 80.

ESSENTIAL REQUIREMENTS FOR A GOOD ORTHOPTIC DEPARTMENT*

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In establishing an orthoptic department, first at the Milwaukee Children's Hospital, and later in expanding and developing the private patient's clinic at the Milwaukee Ophthalmic Institute, it was the intention of the Milwaukee ophthalmologists concerned to do all in their power not only to make an effort to bring about cosmetically straight eyes in the strabismic patient, but, in addition, to supply the means whereby the brain could be reëducated to permit the eyes to function as one complete and single unit in daily life.

Orthoptic training is definitely not a cure-all for the strabismic patient and should not be looked upon as a last resort by the ophthalmologist. We do not consider it a substitute for any other form of treatment but rather as an adjunct to these. Granted that in some instances the patient may dispose of his previously prescribed glasses or may avoid surgery, it is nevertheless, as stated previously, the principal purpose to teach or reëducate the brain to permit the two eyes to function as one single unit by use of the various means available. During this process, which may take from 6 months to 12 or 18 months or even longer, orthophoria may also develop.

When first establishing the orthoptic clinic at the Milwaukee Children's Hospital on a full time basis, we requested a sufficient amount of space to permit of a quiet and undisturbed workroom. This allowed the patient and technician to become acquainted in a gradual and unhurried manner, the attention of the patient being unimpeded by outside disturbances. There are arguments both for and against having more than one technician and patient working in the same room simultaneously. We have found that our results at the Institute where we deal with several private patients in the same room at one time compare favorably with those at the Clinic where only one technician is present at a time. Sufficient space must, however, be allowed so that a certain amount of privacy can be maintained. Screens, simulating booths, between the instruments help to give this impression of privacy. A certain amount of beneficial competition can also be stimulated where more than one patient is present for treatment during each appointment. Frequently the shy and timid child gains considerable confidence from seeing another child present, as it is our habit not to have the parents in the room with the patient during the treatment period. Time is allowed for the parents before and after the treatment for questions, and the like.

In establishing a unit of this type it must of necessity be controlled by ophthalmologists who are in sympathy with orthoptic training and who understand the fundamentals involved in this work. The technician works under the supervision of the ophthalmologist and must be able to carry out his instructions intelligently.

Inasmuch as the technician who has been certified by the American Orthoptic Council has subscribed to a code of ethics which prohibits her from carrying out any treatment not ordered by the attending ophthalmologist, close cooperation and consultation on the part

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*Read before the fourth annual Symposium on Orthoptics, at Chicago, October 10, 1943.

of both physician and technician are required in order that the maximum of value may be obtained. As can readily be seen from the foregoing, it is imperative that the ophthalmologist be familiar with such phases of the work as the accepted and approved treatment for an abnormal retinal correspondence. It would prove rather embarrassing, for example, if home training on a Holmes stereoscope were ordered in this type of case.

The association between ophthalmologist and technician is a close one and one in which mutual confidence must be felt. Because the ophthalmologist customarily leaves the mechanical phases of the procedures to be followed to the discretion of the technician, it is imperative that she be well trained. She must be capable of holding the interest of her charges, most of whom are children. She should have a basic understanding of educational psychology and child management. She must also have an unlimited amount of patience as well as intelligence, a keen imagination, and a sense of humor.

She must have an intense interest in and respect for children as personalities and should derive real pleasure from their association. Above all, unless the patient and parents (provided that the patient is a child) have complete confidence in her ability, the technician will find her usefulness greatly curtailed and the best results will not be achieved.

It is the technician's responsibility to familiarize herself with the various types of equipment that are available and most beneficial in each individual case and to apply from her training the various types of treatment indicated. The routine work which after a period of months becomes rather tedious and boring to the patient must be glamorized as much as possible by the trained technician.

Assuming that the technician is a properly qualified individual a careful selection of patients must be made on the bases of type of squint and personality. Patients having a paralytic strabismus of obvious organic pathology, or Duane's syndrome, are not accepted for treatment. If more than one technician is available the patient is assigned to the one most likely to obtain and maintain coöperation. We make it a policy, however, to rotate the patients to a certain extent, for such rotation helps create new interest and stimulus for both technician and patient. We find it more satisfactory also for our periodic discussions, if this is done relative to the type of work to be carried on, or the advisability of continuing with orthoptics.

The parents of children under treatment must accept certain responsibilities. In our original conference with the parent we discuss the patient's disposition, traits, and general characteristics, and lay out a tentative course of treatment. It is explained that functional training is an educational process just as is learning to play the piano and that time must be allowed as in orthopedic work and in orthodontia. It requires just as much attention, cooperation, and practice. Responsibility for much of this must rest upon the parent. Patients are seen by appointment, for which a definite period is reserved either twice or three times a week for an approximately 30- to 40-minute working period. Parents who show a negligent attitude toward keeping their appointments regularly or promptly are quickly called to task. Shortening of the allotted working period will usually bring the desired results as even though the charges are reasonable the fact that the period of training covers so many months makes it an economic factor of importance in most homes in our locality.

When a patient fails to make satisfac-

tory progress a private conference is held with the parent. The child is also given the responsibility of coöperating in a more satisfactory manner. In the Clinic at the Children's Hospital, we are occasionally able to call on the social-service department to make a home call, although usually, when this is necessary, the parents are neither mentally nor emotionally able or willing to accept responsibility. Frequently by contacting the school, either directly or through the parent, more can be accomplished toward better coöperation, for the lack of proper understanding in the school of what is being aimed at is an important factor in the patients' poor response to treatment.

The terms "transferred" and "referred" have been discussed as to which is the more applicable when used for the procedure we follow.

In the Milwaukee Children's Hospital clinic the strabismic patient is sent or referred to the eye department. Here a preliminary diagnosis is made, a retinoscopy is performed, and glasses, if necessary, are ordered. The patient is then referred or transferred to the orthoptic clinic.

After a series of diagnostic orthoptic tests have been made by the certified technician, the ophthalmologist in charge of the orthoptic clinic decides whether or not orthoptic training will be of any value or if an attempt should be made merely as an experiment. This can be done more easily as an experiment in a clinic than with private patients. Following the conference of the ophthalmologist and the technicians the question of occlusion and a course of training are decided upon. Patients in the orthoptic clinic at the Hospital return periodically to the doctor in charge of the orthoptic clinic or as often as he wishes to see them. After the orthoptic department has decided that surgical intervention should be considered he is "transferred" or "referred" back to the original referring ophthalmologist, who either performs the surgery himself or else arranges to have it performed by another ophthalmologist, usually the one in charge of the orthoptic clinic. In other words, the orthoptic clinic controls the length of time that uninterrupted treatment is to be carried on, but the original ophthalmologist performs the surgery if he so desires. In this manner the orthoptic department cannot be exposed to the charge of "stealing surgery." All other phases of the work as occlusion, reducing the strength of the lenses, or removing the glasses entirely, are under the control of the orthoptic department.

A similar procedure is followed at the Institute. Patients are referred or sent to us by the ophthalmologists on our staff and provided space is available also by any ophthalmologists in good standing with the Milwaukee County Medical Society. Written permission for occlusion and other treatment is sent to us, and a form filled out containing all the necessary data regarding the fundi, refraction, and type of squint. As stated previously, we do not accept certain types of cases such as paralytic, and others. The first visit is considered more of an introductory visit than a treatment, thus permitting the child to adjust himself to us and accept us as friends. As stated previously, patients are seen two or three times a week by appointment. It is our custom to carry out a series of diagnostic tests covering five or six visits to the Institute. Following this, a complete report is sent to the ophthalmologist, who then discusses the various factors involved with the parent or patient, depending on the age. Provided that the patient returns for active orthoptic training he is then given intensive *supervised* training at the Institute and when possible some additional home work is also given. We feel very definitely that only the exceptional individuals may work

at any instrument without constant supervision. Even in the adult, eyes and attention will wander when the patient is not supervised. Our patients at the Institute return to their ophthalmologist periodically every two or three months, as may be desired. The attending ophthalmologist regularly confers with the technicians in regard to reducing the strength of the lenses or when surgery is indicated.

We have accepted patients both at the Institute and at the Clinic for orthoptic training where for economic reasons or others equally good it was not deemed feasible to try to continue with training until single binocular vision could be established. Rather orthoptic training was continued only until such a time as equal vision and a normal retinal correspondence with some fusion amplitude was developed before cosmetic surgery was performed. This policy may not be approved in all areas but it does solve the problem for the patient with barely average finances.

Because of the length of time involved until the course of treatment is brought to a close it is important that fees be kept at a minimum. They must, nevertheless, be sufficient to maintain the clinic or office on a self-supporting and paying basis. In order to eliminate any "dead" accounts from among our active patients we have followed the policy of requesting that payments be made in advance, either on a monthly or semimonthly basis. As a result, our financial losses to date have been negligible. A standard fee is set for a diagnostic series and for a monthly series with two visits per week, and also with three visits per week. Single visits which are used for periodic return visits only also have a set fee. No money may be refunded, no rebates are granted, and no professional courtesy is extended. The patient is instructed in all these points before beginning treatment. We therefore feel that even though the technicians must discuss the question of payments with the patient as well as notify him when each new series begins, the situation is less annoying or embarrassing than would otherwise be the case. The ophthalmologists sponsoring the Institute have been instructed to refer only those patients to the Institute who are able to carry the additional expense of these treatments. The Children's Hospital Clinic assumes the care of the majority of those unable to attend the private clinic.

As stated previously, quiet and privacy are necessary requisites in choosing a workroom. A separate room must be supplied as a reception room to eliminate all possible disturbances. If possible, the working space should be 20 feet in length, to permit carrying out examinations and treatments requiring optical infinity.

Certain equipment must be considered as essential whereas other types may be classed as accessory or supplementary equipment only and therefore not an absolute necessity.

The following equipment is necessary for the diagnosing of a muscle imbalance and type of strabismus satisfactorily and completely:

1. Muscle light at 20 feet and 33 cm. Accommodation chart at 20 feet and 33 cm. for determining an increase in the angle of deviation when accommodating (33 cm. is the distance used in our two clinics for all near measurements).

2. Paddle for alternate or single cover test.

3. Set of loose prisms varying in strength up to 50 p.d. (It is advisable to avoid the very small square prisms, for they are more difficult to hold. The plastic sets avoid the constant danger of breakage but have the disadvantage of scratching easily. A rotary prism, or prism rack is also desirable but not a necessity.)

4. Some method for determining the

type of fusion present in daily life or lack of fusion such as the Worth Dot Fusion Test for both 20 feet and 33 cm.

5. A visual acuity chart as well as an illiterate chart.

6. Some means for testing for amplitude of accommodation and near point of convergence.

7. Adequate records for keeping data on each patient on all visits. In addition to these items some ophthalmologists prefer the Priestley-Smith tape or the perimeter, although we have found the cover test with prisms the most satisfactory method for measuring the angle of deviation.

8. A major amblyoscope, such as the synoptophore, synoptoscope, and orthoptoscope, is an absolute necessity for diagnosing, reëducating the brain, and developing good fusion ability, especially when an abnormal retinal correspondence exists. The first two instruments mentioned are of English design and at this time almost impossible to obtain. All three of these meet the approval of the American Orthoptic Council.

The Wottring Troposcope, which may soon be available as a major amblyoscope, is being planned to meet the approval of the Council as such.

Among the supplementary instruments which meet the Council's approval are the stereo-orthopter and the rotoscope. These cannot be used in all types of cases as can the major amblyoscopes.

Among the more simple pieces of supplementary equipment, a few of which are advisable for variety and interest for the patient, we have the diploscope, the Remy separator, the cheiroscope, the Correct-Eye-scope, and the bar-reader.

The hand diploscope and Remy separator can be made by anyone who can use a ruler and a few tools. Both help the patient in his transition into daily life and stimulates single binocular vision. The cheiroscope and Correct-Eye-scope are intended to teach brain, hand, and eye coördination. The metronoscope with a phorometer is also a supplementary instrument used especially for certain phases of fusion training. The Tel-Eye-Trainer has a mechanical attachment which automatically can control the type of flashing desired. It also is a glorified stereoscope. The Howard-Dohlman apparatus is still an accepted means for measuring depth perception.

The establishing and developing of an orthoptic department is a satisfying accomplishment for both the ophthalmologist and the orthoptic technician. We do not expect to "cure" the strabismic patient, but we hope by one means or another to give him single binocular vision and orthophoria if possible. This is a goal we do not always succeed in reaching but much satisfaction is felt when we do succeed.

Milwaukee Ophthalmic Institute.
Milwaukee Children's Hospital Clinic.

Discussion

Dr. F. Bruce Fralick (Ann Arbor): One of the first essentials in establishing an orthoptic clinic is the local ophthalmologist's honest desire for help from the orthoptic technician. It must be admitted that few ophthalmologists possess full understanding of the subject or the temperament required to carry out these exercises efficiently. This lack of understanding creates a strong feeling of skepticism, places the technician on the defensive, and thus hampers the fullest coöperation between the technician and the oculist.

Secondly, a successful orthoptic clinic can be maintained only by a well-trained technician who has the personality not

only to develop the fullest coöperation of the patients but that of the parents. No amount of basic training will compensate for lack of personality. The best indication of technical ability is a Certificate from the American Orthoptic Council, but recommendations of the technician's sponsors and one's own impression of her personality are most important.

The technician's duties should not include those of the office nurse unless she is permitted so to arrange the time as to give her undivided attention to the patients receiving orthoptic training. Without this arrangement, frequent interruption in her work would result and most likely make it impossible for her to regain the child's interest in the work for the day.

Those contemplating developing an orthoptic clinic would do well to follow the suggestions outlined in the discussion by Miss Dicke.

University Hospital.

PRECAUTIONS NECESSARY IN ORTHOPTIC TRAINING*

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In discussing the technical application of orthoptics too strong an emphasis cannot be placed on the necessity for observing certain essential precautions.

If, in setting up her routine, the orthoptic technician will establish a procedure of checks and balances, possible errors can be reduced to a minimum and the benefits derived will help the patient as well as the technician. At the initial examination, a careful study of the patient's eyes for motility constitutes a primary procedure. Vision with and without glasses should be recorded, and measurements made with prisms and cover-test at six meters and 25 cm. with and without glasses. With full correction and the addition of a +3.00D. sph., the patient is measured at 25 cm. to determine his accommodative factor. A check should be made with red and green glasses to define the type of diplopia present. Worth's Four Dot test is then employed to ascertain if binocular vision exists.

The next step is the Bielschowsky test for the evaluation of the afterimage. The patient's subjective and objective angle is then measured on the synoptophore, and the presence or absence of fusion and depth perception determined. Upon completion of this orthoptic survey, the technician knows the ocular imbalance and the status of binocular vision, and can therefore proceed to treat her patient with a maximum of intelligence in a minimum of time without wasting effort on skills within the patient's scope.

Upon reaching this point, the range of the patient's performance orthoptically being understood, it is extremely important to discuss the case (if the patient is young) with the parents and to infuse both parents and child with a thorough understanding of the coöperation expected during the course of treatment. Lacking this understanding and at least a modicum of enthusiasm, little can be accomplished, and orthoptic treatment, even in the mildest cases, will be ineffective. Since the patient's emotions are involved, there are times when, to secure proper response, a degree of ingenuity and a knowledge of child psychology are as invaluable to the

* Read before the fourth annual meeting of the American Association of Orthoptic Technicians, Chicago, Illinois, October 11, 1943.

technician as her instruments. Another precaution well worth observing is that of treating young patients early in the day before fatigue complicates matters by reducing attentiveness. During all stages of orthoptic treatment, it is essential that the patient be encouraged to concentrate and to accomplish the utmost within the compass of his mental equipment.

If the examination shows the presence of amblyopia ex anopsia, occlusion is necessary. Occlusion may range from a total type to a light application of lacquer. Total occlusion is imperative if the amblyopia ex anopsia is of a marked degree. The precaution of seeing that the occlusion recommended by the technician is properly carried out is important. However, it has been definitely proved that occlusion in itself will not restore impaired vision. Organized and supervised homework for exercise at 6 meters as well as for near is efficacious; if reviewed and checked at intervals by the technician, it proves well worth the effort expended.

If anomalous retinal correspondence is present, total and constant occlusion becomes of vital importance. Homework is not recommended in such cases, inasmuch as it tends to stimulate the abnormal retinal correspondence. The Bielschowsky afterimage test and the major amblyoscope have been found indispensable for checking and treating patients in this category. Particularly during this stage of treatment must the technician give unstintingly of her enthusiasm and resort to her highest skills to sustain the confidence and coöperation of her patient. A thorough understanding of what the technician aims to accomplish reduces the patient's apprehension and helps to obtain his fullest coöperation. After normal retinal correspondence has been established, the patient's condition may be treated as an ordinary fusion case. If the angle of deviation remains the same after fusion has been established and an attempt has been made to develop amplitude, an operation is indicated.

In the treatment of suppression, prevalent in a high percentage of cases, occlusion is employed, its extent being commensurate with the degree of suppression. Again, it is a wise precaution to instruct the parents and child in the reasons for, and the proper use of, this medium. The better their understanding of the technician's goal, the more satisfactory the results. Enforcing the correct usage of occluders presents one of the technician's most difficult problems, hence extreme precaution must be taken to insure the patient's continuous, sincere cooperation in following the prescribed routine.

In uncomplicated accommodative squint, cures have been effected in from two to eight months, depending upon the intelligence, coöperation, and concentrating ability of the patient. Children of school age can be readily taught to dissociate accommodation and convergence. In treating fusion cases in which no suppression or abnormal retinal correspondence is involved, unless improvement is noted within a reasonable period of time, the patient should be referred back to the ophthalmologist.

It has been our good fortune that most operative cases treated prior to surgery have been returned to us by the ophthalmologist for postoperative treatment or checkup. The precaution of checking the patient at intervals after proper muscle balance has been established might be stressed at this point. In a large percentage of our cured strabismic cases, the ophthalmologist recommends a yearly examination. Recently, it has been our privilege to check several patients, who, after a cure of six years' standing, showed no deviation whatever and enjoyed comfortable binocular vision. What greater en-

couragement could a technician desire?

In concluding, I wish to mention that the precautions necessary in orthoptic treatments are so multiple and of such manifold character that only the highlights, the most important precautions have been discussed in this paper; namely,

1. Examining the patient for deviation and binocular capacity.

2. Imbuing parents and child, where occlusion is used, with a thorough understanding of its purpose, and the role they must assume, under the technician's regular supervision.

3. In anomalous retinal correspondence, no homework should be given.

4. If no improvement is observed after reasonable treatment, the patient should be referred back to the ophthalmologist.

5. Patients should be encouraged to see their ophthalmologist for a yearly check after cure or establishment of normal muscle balance.

Eye and Ear Hospital.

NOTES, CASES, INSTRUMENTS

A METHOD FOR THE PROJECTION OF EYE SPECIMENS*

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Saint Louis 3

A simple and convenient method for the projection and demonstration of sectioned pathologic material is desirable not only in presenting case reports, but also in the routine teaching of pathology. In most specimens in general pathology a relatively high magnification is necessary to give some histologic detail. In the study of ocular pathology the majority of the important facts can be demonstrated with a relatively low power. In fact, the relationship of intraocular pathology can often best be shown with lower magnifications than are available in the usual microprojectors. The usual apparatus available is a modification of the ordinary microscope with a powerful source of illumination, usually a carbon-arc. These instruments are expensive, complicated to operate, and relatively stationary. The following method for the projection of eye specimens has proved to be simple, cheap, and always available.

A celloidin section of a globe that has been cut in the ordinary manner is stained, usually with hematoxylin and eosin. Instead of mounting on a microscope slide with cover slip, the stained section is mounted in gum dammar on a 2 by 2-inch lantern slide. A similar slide is used as a cover slip and the edges carefully approximated. The excess gum dammar is expressed and the slide allowed to dry for several days. The edges are then bound with lantern-slide tape. Personally, I have never mounted a paraffin section in this way, but I can see no reason why this type of slide could not be substituted for the ordinary microscope slides. This slide can then be projected on any of the small 2 by 2-inch projectors, such as are used for kodachrome transparencies. In the ordinary room the image of the eye on the screen is about three to four feet in diameter. Such gross lesions as tumors are best shown. If it is desired to show any histologic detail, this can easily be done with the same projector, using 2 by 2-inch photomicrographs.

This method seems quite obvious, but I am reporting it because I have never seen it described, and it has proved interesting to a large number of ophthal-

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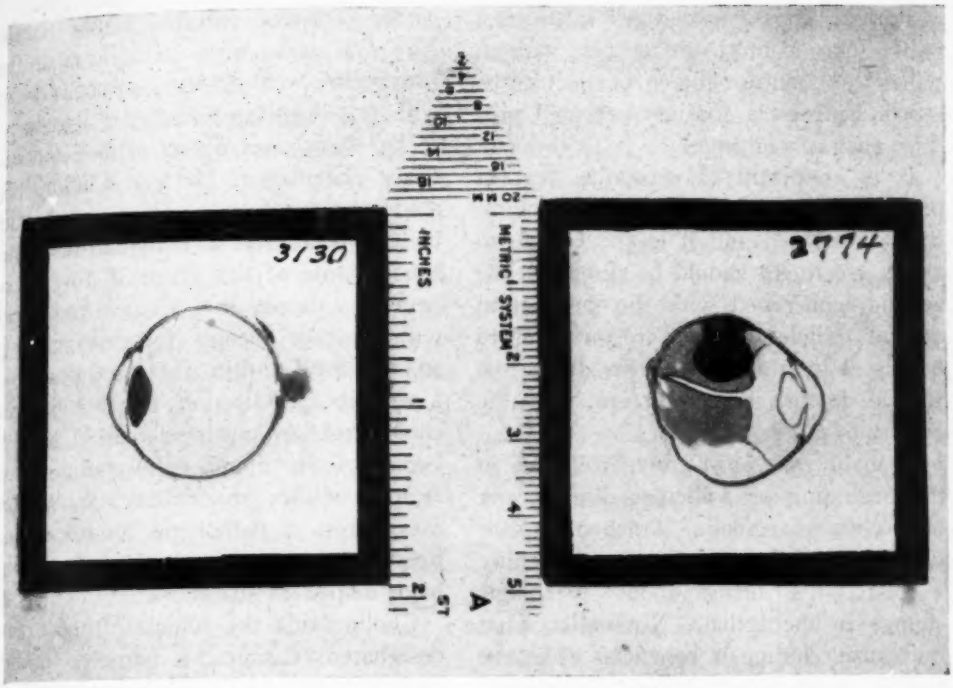

Fig. 1 (Sanders). Projector slides prepared in the described manner, showing a globe with glaucoma and one with tumor.

mologists, none of whom had seen it previously.

508 North Grand Boulevard.

THE TREATMENT OF TRICHIASIS WITH CHOLINE*

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As regards trichiasis, clinicians are agreed upon the definition that it is an affection of the cilium and primarily its follicle. As a consequence of the disease of the hair follicle, the cilium is poorly nourished and turns inward in a lifeless manner to impinge upon the cornea.

Although trichiasis pathology is recognized as an eyelash-follicle disease, such knowledge is not put to use in its treatment, for when epilation and electrolysis alone are adopted, no consideration is given to the disease in the follicle. The effects of these two means are of but short duration, for the lashes continue to develop from the site of the follicle supposedly destroyed by electrolysis.

At the University of Salerno, in the tenth century, a different approach to this problem was approved. Bile was instilled into the eye to cure the disease in the follicle. Though the use of bile is legendary, according to the historians of today, we propose to show that choline, a constituent of bile, is a most remarkable agent for the treatment of trichiasis.

There occurs at times an aberration in lid development in which the tarsus is absent or undeveloped and the lashes unsupported, are left to strike the cornea; but this is not trichiasis in that the follicle

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is normal. It is a recognized fact that a cilium may contact the cornea without either the cilium or the cornea being diseased. Entropion, for instance, will produce such a condition.

It is important to recognize that in pathology of trichiasis, it is the follicle that is diseased and it is the follicle to which treatment should be directed. We are not concerned with the destruction of the follicle but with restoring it to health. We aim to remove from the follicle factors that interfere with the growth of a normal cilium.

Probably the most common factor in the beginning of follicular disease is a blepharitis marginalis. Trachoma, pemphigus, and deficiency diseases that cause a blepharitis bring about pathologic change in the follicle. No matter what the cause, choline is beneficial when the follicle is filled with lipoids and cholesterol.

The cilium has its root some two to three millimeters within the follicle and is nourished by the glands and vessels about the follicle. Its curve depends upon the base of the root and the exposed length as, also, upon its shape and diameter. Near the lid border the cilium is cylindrical. Farther out from the lid margin the cylinder flattens and becomes narrowed and, because it lacks equal support throughout its entire extent, it curves. In trichiasis there is no longer the normal curve; the cilium falls as though from a physical inertness.

Choline, which we have found so beneficial, has been fully described in biochemical, physicochemical, and physiologic treatises. It has been well established as a lipotropic agent of great merit. It is because of the lipotropic power that we adopted it in the treatment of trichiasis.

The pH of choline in aqueous solution, 5 grains to the ounce, is nearly perfect for the external ocular tissues (7.1). It is so little irritating that the conjunctival cul-de-sac may be flooded with it almost continually, if necessary. *It has been used by us to lave the anterior chamber of the eye.* Choline has moderate antiseptic qualities and, having both a polar and a nonpolar molecule, can, because of this chemical formation, penetrate deeply into tissues and carry with it other agents, if necessary. It is soluble in oil and in water and possesses a definite predilection for lipoids and cholesterol; so much so that in the reversal of the lipoids, phospholipids are formed which are readily transported away from a pathologic condition that previously existed, and which they may have helped to maintain.

Choline rids the follicle of these two designated chemicals; namely, lipoids and cholesterol. Besides possessing all of these unusual qualities, *it seeks out diseased channels to travel in and through in order to reach a pathologic condition.*

In trichiasis choline is employed locally either in water or in an oil solution and may be used in crystal form directly applied to the lesion in need of treatment. In water and in oil it is used in a strength of 5 grains to the ounce. In whatever manner it is used, it penetrates into the follicle. It may even be used orally to affect the trichiasis, but the treatment is extended.

We have not and do not expect to observe any harmful effects from its use in the treatment of trichiasis, and our experience has been great.

The time required for effective action in trichiasis is but a week or thereabouts. It is to be borne in mind that choline is also a detergent, and because of this quality can be used in the removal of scabs from the face and lid margins.

Twenty cases of trichiasis have been treated with a solution of choline. Two

of the patients also had pemphigus and one had trachoma. Choline had a remarkable effect in each case. In the case of pemphigus choline was used as an eye wash; in the case of trachoma, crystals of choline were applied to the lids.

In conclusion: The lipotropic agent choline is most remarkable in its effect upon trichiasis. The simple trichiasis was selected from a series of pathologic changes to demonstrate the basic potency of choline. It chemically removes lipoids and cholesterol from diseased follicles without altering the normal follicle. It appears to restore the follicle to health. New lashes may be formed normally where the diseased lash previously existed and it accomplishes such action without harming tissues.

A CASE OF INTRALENTICULAR FOREIGN BODY WITH EARLY REMOVAL*

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A case of intralenticular foreign body is presented.

A. De G., a man, aged 39 years, on March 18, 1942, while hitting a chisel with a hammer, felt a piece of steel strike his right eye.

Examination revealed the following findings: Vision without correction: O.D., 20/70; O.S., 20/20. Vision with correction: O.D., 20/40 plus 2; O.S., 20/20. The patient was wearing O.D., −0.75D. sph.; O.S., −0.50D. sph.

External examination showed a linear scar on the cornea, which penetrated vertically through its entire thickness. The anterior chamber was intact, and on the anterior capsule of the lens there appeared to be a small linear opacity, which under the slitlamp was seen to extend into the nucleus. A small glistening object, thought to be the foreign body, was seen. Ophthalmoscopy revealed an opacity of the lens. The patient was admitted to the New York Eye and Ear Infirmary. X-ray studies localized a radio-opaque foreign body in the lens. On March 20th, the foreign body was removed by means of a giant magnet. It was first brought into the anterior chamber by applying the magnet to what had been the path of entry. The anterior chamber was opened at the limbus with a keratome and the foreign body brought out through the incision with a hand magnet.

The patient was put on atropine, vitamin-B complex, and vitamin C, and treated for a month. By this time the eye was white and with −0.50D. sph. ⌯ −0.50D. cyl. ax. 75° the vision was 20/30. One year later, on April 1, 1943, the right eye with −1.25D. cyl. ax. 75° still had 20/30 vision.

Two of the most important arguments in favor of the early removal of intralenticular foreign bodies are (1) the prevention of complete opacification of the lens, and (2) the prevention of siderosis bulbi. It is commonly thought that when lenticular foreign body is diagnosed, it is best to wait until the traumatic cataract has become mature and then remove the foreign body along with the cataract. Frequently, when the anterior capsule of the lens is injured, the aqueous is absorbed by the lens fibers, which then become swollen and disassociated, forming a traumatic cataract. In some cases, however, if the foreign body is removed early, the torn anterior capsule will coapt quickly and heal without permitting the aqueous to penetrate the lens fibers. Moreover, the presence of a metallic foreign body in any part of the eye leads to the deposit of iron pigment throughout the entire eye. The prevention is early removal.

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*Read before the New York Society for Clinical Ophthalmology on February 7, 1944.

This case is presented as a demonstration of the advisability of early removal of lenticular foreign bodies. In this instance, the anterior capsule had been ruptured and the foreign body had lodged in the lens. Early removal of the foreign body resulted in the formation of a localized lenticular opacity only. The lens did not become completely opaque. There was no evidence of siderosis bulbi. The visual result was extremely satisfactory. The patient is able to see 20/30 with correction and has binocular vision.

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PENICILLIN IN THE TREATMENT OF PURULENT CONJUNCTIVITIS

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Since penicillin is not secreted in the tears,[1] one cannot expect its full effectiveness in conjunctival infections by either intramuscular or intravenous administration. The Oxford group,[2] in 1941, reported four cases in which sodium penicillin was used as drops into the eyes with excellent results. In 1943, Florey and Florey[3] treated a small series of cases using calcium penicillin as drops and ointment, with good results.

With these facts in mind, as well as the limited supply of penicillin, one case of purulent staphylococcus conjunctivitis was treated with local instillation of sodium penicillin as drops, and is herewith reported.

R. V. M., a male infant, aged 25 days, born six weeks prematurely, was first seen at home on March 6, 1944. The mother stated that a profuse purulent discharge in both eyes had begun a day after birth. During the last 24 days, the child had been treated, at first with hourly irrigations of boric acid and argyrol. Two unknown drugs were also used in the eyes during this period. During the last week, the eyes were irrigated only every three hours, but the lids were always adherent with purulent discharge after 30 to 60 minutes. The general condition was fair considering the prematurity of the patient.

Examination. All four lids were congested, swollen, and sealed shut with a thick, yellow, purulent discharge which had formed an external dry crust. On opening the eye, a large quantity of pus ran out of both eyes. The conjunctiva was extremely congested, but the cornea of each eye was clear. A smear made from the discharge revealed gram-negative, biscuit-shaped, intracellular diploccocci with a few gram-positive cocci.

The infant was sent to the City Isolation Hospital for treatment. On admission, the culture made from the secretion from the eyes revealed hemolytic and nonhemolytic Staphylococcus and hemolytic and nonhemolytic Streptococcus. The general examination was negative, except that the patient was quite emaciated, weighing 5 pounds and 10 ounces.

Treatment. The eyes were irrigated with a saturated solution of boric acid every half hour, followed by several drops of 5-percent sodium sulfathiazole solution. Sulfathiazole was given orally, 2 gr. every six hours.

On March 9, 1944, no gonococcus was found by smear or culture. The culture was positive for only hemolytic Staphylococcus. The discharge was still profuse, requiring irrigations and sulfathiazole at half-hour intervals.

Nine days later, because there had been no apparent progress in this case, the sulfathiazole was discontinued. Cultures were still positive for hemolytic Staphylococcus. The patient was skin tested with staphylococcus antitoxin and was found

to be very sensitive to it; therefore no antitoxin was given.

On March 20th, the discharge was not quite so marked, necessating irrigation at hourly intervals to keep the lids from adhering. The latter were congested and swollen and the conjunctiva markedly congested. A solution of sodium penicillin in normal saline containing 700 Florey or Oxford units per cubic centimeter was prescribed for use as local treatment, two drops of the solution being instilled into each eye every hour, after the eyes had been thoroughly irrigated with a boric-acid solution. A culture positive for hemolytic Staphylococcus was obtained from each eye at 6:00 p.m., and the medication was started at 7:00 p.m. Cultures were ordered every six hours before the eyes were irrigated.

On the following day, at 12:00 a.m., the cultures taken after five hours of treatment with penicillin were positive for hemolytic Staphylococcus in both eyes. The 6:00 a.m. cultures from both eyes were positive for hemolytic Staphylococcus, but the discharge had practically ceased. The 6:00 p.m. culture was negative and there was no discharge, so irrigations were discontinued, but the instillation of penicillin was continued every hour.

On March 22, 1944, at 12:00 a.m., one colony of hemolytic Staphylococcus was found in the culture from the right eye; the culture from the left eye was negative. All subsequent cultures, taken at six-hour intervals until March 24th, were negative. In other words, there was only one colony after 17 hours of treatment, and no positive cultures 24 hours after the treatment with penicillin was initiated.

On the following day at 12:00 noon, the administration of penicillin was stopped. The eyes appeared normal and were normal at the time the infant was discharged from the Hospital, on March 26th.

A disadvantage in using sodium penicillin is that it is not very stable, but we had the good fortune of having a fresh solution prepared every few hours for another patient who was being treated at the same time, thereby keeping a fresh supply for our patient. Calcium penicillin is more stable and would be preferable for local treatment of conjunctival or corneal infection.

The successful treatment of one case does not prove anything, but the results were so spectacular in this case, which had been resistant to other treatments, that we felt it worth reporting.

We are indebted to Mrs. Dorothy D. Seely for her work on the bacteriology in this case and to Dr. Blaine Z. Hibbard for his constant observation of the patient.

References

[1] Rammelkamp, C. H., and Keefer, C. S. The absorption, excretion, and distribution of penicillin. I. Clin. Investigation, 1943, v. 22, pp. 425-437.

[2] Abraham, E. P., Chain, E., Fletcher, C. M., Gardner, A. D., Heathley, N. G., Jennings, M. A., and Florey, H. W. Further observation on penicillin. Lancet, 1941, v. 2, p. 188.

[3] Florey, M. E., and Florey, H. W. General and local administration of penicillin. Lancet, 1943, v. 1, March 27, p. 387.

SOCIETY PROCEEDINGS

EDITED BY DR. DONALD J. LYLE

COLORADO OPHTHALMOLOGICAL SOCIETY

May 15, 1943

DR. C. A. RINGLE, *president*

DOUBLE HYPERPHORIA

DR. GUY HOPKINS presented the case of A. A. M., aged 34 years. This patient was first seen on November 19, 1941, during a routine examination for an aviation certificate.

The vision in the right eye was 20/25, improved to 20/15 with −0.25D. sph. ⊂ −0.50D. cyl. ax. 65°. The vision in the left eye was 20/20, improved to 20/15 with −0.25D. sph.

Examination of the muscles showed a left hyperphoria of 8 prism diopters with the Maddox rod, but by means of the cover test a right hyperphoria of 6 degrees and a left hyperphoria of 6 degrees were found. The patient had diplopia in the upper right and upper left temporal fields. The right hyperphoria increased on looking up and to the right. The findings were not constant and varied from time to time.

SPONTANEOUS HEMORRHAGE FROM IRIS INTO THE ANTERIOR CHAMBER OF THE RIGHT EYE

DR. GUY HOPKINS presented the case of Miss B. C., aged 48 years. She was first seen on April 7, 1943, and complained of a hemorrhage into her right eye with a history of having experienced one year previously a similar attack which occurred after stooping.

Vision in the right eye was 20/80, corrected to 20/25; in the left eye it was 20/60, corrected to 20/20. Examination revealed a blood clot in the anterior chamber of the right eye extending from the middle of the lower portion of the iris, on a line from the 12- to the 6-o'clock position, upward and on to the anterior lens capsule. The pupil was not dilated at this time.

On April 12th the pupil was dilated, and the iris showed one small posterior adhesion at the 12-o'clock position which was easily broken up with atropine. Examination of the eye was otherwise negative for any pathologic change.

Physical examination was essentially negative. The patient had had a cholecystectomy in 1939. Since that time she had taken bile salts by mouth. She showed some yellowness of the scleras. Her icterus index was 24, coagulation time 35. She had been given Bilron and vitamin K. The hemorrhage entirely cleared up within a week, and the patient suffered no recurrences.

COMPLICATED CATARACT OF THE RIGHT EYE

DR. GUY HOPKINS reported the case of G. B., aged 68 years. He was first seen on November 25, 1925, at which time he had a fully developed cataract in the right eye. Vision in the left eye was 20/20. When he was seen again, several months later, there were a few small deposits on Descemet's membrane. Since that time he had been seen at intervals through the years because of various minor complaints and for changes in his glasses. The left eye always remained normal.

On July 18, 1942, he returned, stating that the cataract in the right eye had become more noticeable. The pupil was dilated and fixed. The iris was atrophic, and the lens was dislocated. He was

placed on pilocarpine and observed. In August, 1942, he returned with an acutely inflamed and painful right eye. He had accidentally struck his eye the preceding day.

Examination revealed secondary glaucoma. The tension was 60 mm. Hg (Schiötz), and the anterior chamber was filled with blood. Under rest and miotics the tension returned to 26 mm. Hg and remained at about this level except for two very slight flare-ups.

Intraocular foreign body

Dr. C. E. Earnest presented the case of C. P., aged 26 years, who gave a history of having sustained an injury to his right eye about two years previously. The patient reported for examination upon his discharge from the Army with a diagnosis of a piece of steel in his eye. X-ray examination of the eye was made and an extracapsular lens extraction performed. The rusty area in the cataract was then tested with a magnet and found to be magnetic. This particle of steel, however, was so small that it was absolutely impossible to remove without extracting the lens. A marked siderosis was present.

This case was reported because it showed how an extremely fine piece of steel can be missed even with X-ray examination, since there are many metals that do not show in X-ray pictures.

Ocular injury

Dr. C. E. Earnest reported the case of M. I. S., a civil engineer, aged 36 years. On February 21, 1935, while inspecting in an ore mine, dynamite exploded, injuring his eyes and face and knocking him into a hole, 10 or 15 feet deep. He had a traumatic cataract of the right eye, with multiple lesions of the cornea and iris, and fine particles of ore in the anterior chamber, posterior chamber, and vitreous. A cataract extraction was performed and, with correction, the patient was able to resume his regular work, even though many particles of ore remained in the eye. This case was reported because ore does not show on X-ray pictures and also because ore is nontoxic.

A number of other cases had also been observed previously. One patient, seen about 13 years ago, had sustained several injuries with tattooing of the cornea and multiple particles throughout the eyeball. He had had cataract extraction and iridotomy, and, with correction, vision was 20/100. The patient resumed his work although he still had particles of ore in his eye. The vision in the left eye was 20/20.

Possible ocular tuberculosis in old injured eye

Dr. C. E. Earnest presented the case of Mr. P. J., aged 28 years, who was first seen on September 11, 1941. He stated that many years ago he had suffered an injury to his left eye. Since that time vision of this eye had been impaired. In the past few months vision in the eye had rapidly decreased. The right eye was normal, vision 20/17. The left eye was slightly reddened, and there was some scleral injection. The cornea was clear, but there were numerous precipitates on the posterior surface. The iris was not congested. The pupils were equal in size, measuring about 4 mm. in diameter, and reacted well to light and accommodation. There were, also, numerous small gray nodules seen with the slitlamp. The lens was clear. There were numerous opacities in the vitreous. The disc was apparently normal but indistinct, and extending into the vitreous was a semitransparent body.

Above the level of the upper margin of the disc there was a whitish mass, practically without prominence. The visual

fields, transillumination, and tension were normal. There was also an old lesion of the lung suggestive of tuberculosis. A tuberculin test was negative.

On September 23, 1941, the left eye was fairly quiet. The posterior surface of the cornea was almost covered with precipitates. There was an exudate along the margin of the pupil. The fundus was very indistinct and the opacities in the vitreous were more numerous. The vision was the ability to count fingers at 10 feet. X-ray treatment was given to the left eye.

During the next six months X-ray therapy was continued at various intervals. The eye became quiet, but there were some cataractous changes in the lens. There was no change in the vision.

The diagnosis was probable tuberculosis of the left eye.

Questionable ocular tuberculosis

Dr. James M. Lamme presented the case of Mrs. A. T., a 49-year-old bookkeeper, who awakened one morning with definite loss of vision in her left eye. She had suffered no headaches nor premonitory symptoms. The lids, conjunctiva, and cornea were normal in each eye. The pupil of the right eye measured 4 mm., that of the left eye measured 5 mm., and the reflexes were slightly sluggish. The vision, R.E., was 20/35−1, improved to 20/20 with correction. The vision, L.E., was 20/100, unimproved with correction. There was no objective pathology found in the right eye.

The fundus of the left eye showed edema and petechial hemorrhages around the disc, which was rather indistinct. There was generalized edema of the retina. There was a massive fan-shaped hemorrhage from the superior temporal vein which extended almost to the ora serrata. A presumptive diagnosis of optic neuritis was made.

A thorough physical examination, X-ray and complete laboratory tests, which included blood and spinal-fluid examination, were negative.

The patient was given potassium iodide and theelin, and bed rest was advised. One month later the vision was still 20/100 and the fundus findings were unchanged. When examined again, one month later, the acute process appeared to be subsiding. The patient was allowed moderate exercise and advised to stop taking the potassium iodide. In December, 1942, dental examination revealed five infected teeth, and these were extracted. Examination of the fundus at this time showed a definite exudate surrounding an irregular and obscured superior temporal vein. There were small veils of exudate over the nerve head, and very marked vitreous opacities were present. The patient was advised to resume taking the potassium iodide, and in February, 1943, a definite clearing of the fundus was noted; the vision was R.E. 20/20, L.E. 20/35−2. Two weeks later the patient started working and continued with the theelin injections every second week.

Examination in April, 1943, showed that the fundus landmarks were completely obliterated by recent swelling and massive hemorrhage around the nerve head. Treatment as outlined during the first attack was resumed. These observations prompted a presumptive diagnosis of tuberculosis.

Walter A. Ohmart,
Secretary.

ROYAL SOCIETY OF MEDICINE

Section of ophthalmology

June 11, 1943

Mr. F. A. Juler, *president*

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BAND KERATITIS

MR. O. G. MORGAN presented a case of idiopathic band keratitis in an otherwise perfectly healthy pair of eyes. The patient was a man, aged 76 years, who gave a history of a gradually increasing corneal opacity for at least six months. There was no evidence of any intraocular inflammation or degeneration. In the right eye vision was less than 6/60, and the band extended across the pupillary area. In the left eye there was a small notchlike clear space just in the pupillary area through which he obtained 6/18 vision. He had not at any time been engaged in work which might have produced a chronic irritation of the eyes. As the disease was in Bowman's membrane and beneath it, optical iridectomy in the poorer eye would seem the most satisfactory form of treatment, especially if the visual acuity in the better eye continued to fail.

Discussion. Mr. Humphrey Neame said that when such a band was definitely calcareous he had found improvement by curetting.

Mr. F. A. Juler said that he had had two or three of these cases which had improved by curetting the central area.

Major E. F. King asked whether a corneal graft had been considered.

Mr. Maurice Whiting said that it might be worth while considering the question of performing tarsorrhaphy in the better eye. Exposure of the cornea in the palpebral aperture had an important effect in the production of band opacities, and if a tarsorrhaphy were done in this case it was possible that the opacity might be prevented from becoming more extensive.

Mr. J. H. Doggart said that he had observed several cases of bilateral band-shaped keratitis not associated with any inflammation, and those which were scraped very soon relapsed. The only two procedures which might help in this case seemed to be (1) an optical iridectomy, and (2) a corneal graft.

Air-Commodore P. C. Livingston wondered whether these cases had any nutritional element in them. It seemed worth while giving large doses of vitamin C intravenously. In somewhat smilar cases he had seen doses of 500 to 1,000 mg. result in improvement. For some unexplained reason the substance seemed to reach the cornea in a more beneficial form by intravenous injection.

Mr. O. G. Morgan, in closing, said that he was quite certain that this condition involved the cornea more deeply than the epithelium. He said he might try vitamin C intravenously, since it could be done fairly quickly, but he felt that iridectomy was the most satisfactory treatment in this case.

BILATERAL BUPHTHALMOS WITH CONGENITAL ANOMALIES OF IRIS AND SUBLUXATED LENSES

MR. V. B. PURVIS presented A. B., a 23-year-old male laborer who had bilateral buphthalmos along with a most unusual condition of the iris, corectopia, and iris dehiscence, with subluxation of lenses.

The patient first appeared at the hospital one month ago complaining that his vision had been defective since birth but had become progressively worse during the last six years. He also stated that for the past few weeks he had been seeing rainbows. Examination showed that the cornea of each eye was larger than normal. He had a moderate amount of myopia and wore −8.00D. sph. lenses. The vision in the right eye was reduced to the perception of hand movements, and in the left eye with glasses to 5/60.

The tension in each eye was elevated to 50 mm. Hg (Schiötz). The corneas were clear and there were only one or two ruptures of Descemet's membrane in each eye. The discs were grossly cupped and pale. The visual field in the right eye was impossible to chart because of loss of fixation, and in the left eye was reduced to within 5 degrees of fixation with a 10-mm. object at 330 mm.

The main points of interest in this case were the ectopic pupils, which were displaced in an unusual direction down and out, and the splits in the mesoderm of the iris. Only one of these splits (in the right eye) was complete, in that there was an accompanying tear of the ectodermal layer of the iris through which a red reflex was obtainable. The condition was not one of true polycoria in which the accessory pupils each had their own individual sphincters.

The case has not responded to eserine therapy and it was decided to perform an iridencleisis in each eye.

Detached retina, questionable cause

Mrs. V. M. Attenborough reported the case of a four-year-old boy who was first seen in February, 1943. He complained that for the past two days he could not see with his right eye. Four months previously he had had whooping cough. The mother stated that he had also sustained a fall some time ago. Examination revealed a retinal detachment of the right eye with what appeared to be a disinsertion from the 3- to the 6-o'clock position. The eye was quiet. There were no keratitic precipitates, no synechiae, and no obstruction to transillumination.

Discussion. Mr. R. A. Greeves suggested that this was a case of Coats's disease. In various places behind the retina there were areas composed of a dense, yellowish-white substance, the appearance of which suggested massive exudates. The vitreous was clear and this, in his opinion, excluded the diagnosis of an inflammatory condition. Detachment of the retina, such as was present here, is common in the later stages of Coats's disease. He would not advise excision.

Mr. Humphrey Neame proposed an alternative diagnosis; namely, a rather unusual endophthalmitis, with the retina very much detached. In one such case, in which the eye was excised, the retina in the posterior part of the eye was pulled forward very much by the tissue strands in the vitreous.

Mr. O. G. Morgan said that because the chance of retention of this eye in the future was very doubtful and, since the pathology was certain, he would recommend enucleation. He cited a similar case in which he eviscerated the eye. It turned out to be a sarcoma and the child died of extension some months later.

Progressive partial thrombosis of the central retinal vein

Mr. P. D. Trevor-Roper (on behalf of Mr. P. G. Doyne) presented the case of a woman, aged 36 years, who in November, 1942, saw black spots in front of her left eye, lasting about one hour. In May, 1943, she noticed black spots again, followed by slight mistiness which gradually disappeared during the next few days. On admission to Moorfields on the next day the right eye was found to be normal and the vision was 6/5. In the left eye the vision was 6/5 and the eye was normal except for the fundus, which showed dusky and engorged veins and a swollen and blurred optic disc. There were two minute hemorrhages along the inferior nasal vein and one near the macula. A course of heparin injections

was given and seven days after admission the patient was discharged. The left eye showed the same condition of the veins as when first seen; increased swelling of the disc with several small hemorrhages around it, and absorption of the small hemorrhages along the inferior nasal vein. When seen on June 1, 1943, the hemorrhages had increased and the papilledema had become more marked. There were at this time about four small hemorrhages around the macula and a dozen scattered along the retinal vein.

A Wassermann test was negative. X-ray examination of the chest was negative. Physical examination showed no abnormality except rather low blood pressure (100/80) on separate occasions, and dental sepsis.

The case was of special interest in view of the slowly progressive nature of the fundus changes resulting from partial central-vein thrombosis; and also in condemning the use of heparin, which was shown to be of no avail even when the thrombosis was in its earliest stages.

Discussion. Mr. F. A. Williamson-Noble said that he thought this was a thrombosis because the slighest amount of pressure on the unaffected eye caused venous pulsation, whereas if one pressed on the unaffected eye there was no venous pulsation until sufficient pressure had been exerted to elicit arterial pulsation, at which time the vein collapsed in the same quick way as the artery.

Chorioretinitis associated with toxoplasma

Col. Derrick Vail (MC), A.U.S., presented a paper on a type of chorioretinitis which is produced by the toxoplasma parasite. A brief history of the toxoplasma was given which included its habitat and host. The method of invasion of the host is unknown at present. Laboratory tests reveal a number of experimental means of transmission of the parasite. A description of the parasite followed.

The infestation affecting the eye was first reported in 1923 by Janku. Since then a few others have reported cases. A résumé of these findings was given.

In the cases described the ophthalmologic examination revealed in both the infantile and adult cases the familiar circumscribed exudative choroiditis. There is a predilection for the macular area, and the lesions are usually bilateral and multiple. Activity of the lesion is characterized by choroidal hemorrhage, usually in the direction of the extension of the lesion. Healing is shown by pigmentation, sometimes accompanied by proliferative retinitis and in one case retinal detachment.

In 11 of the cases described the condition was probably congenital, in the twelfth it was without doubt acquired. Three additional cases occurring in adults past the third decade, under Colonel Vail's observation, have not yet been reported. These are probably acquired cases of toxoplasma.

Iridocyclitis with corneal precipitates has been found twice; vitreous opacities may or may not be present.

The diagnosis in congenital cases can be made by the clinical tetrad of symptoms as described by Sabin. This is: (1) hydrocephalus or microcephalus; (2) cerebral calcification; (3) chorioretinitis; and (4) disturbances of nervous function.

Koch and his associates believe that the intraocular lesions of toxoplasmic encephalitis have certain characteristics that seem to distinguish them from other forms of choroiditis. Among these are, regularity of bilateral involvement of the macular region, tendency to bilateral oc-

currence of other than ocular lesions, presence of massive chorioretinal degeneration, extensive connective-tissue proliferation and heavy pigmentation, tendency to the occurrence of associated congenital defects in the eyes, and constant clarity of the media in the presence of severe chorioretinitis.

However, in Colonel Vail's opinion, there is nothing in the ophthalmoscopic appearance of the lesions that is characteristic, at least in the older children and adults under his observation, provided one accepts the validity of the laboratory test as the diagnostic yardstick.

More studies are needed to prove the validity of the toxoplasma neutralization test. Up to the present the test seems most promising and accurate. Sabin's studies unfortunately have been interrupted by the war, but in a recent article he has described 151 individuals suspected of toxoplasma infection, 59 of whom gave positive neutralization tests for toxoplasma. It is significant that in 9 out of 10 cases in which the sera contained neutralizing bodies for toxoplasma, the only clinical manifestation was that of a chorioretinitis of unknown etiology with a predilection for the macular region. Sabin had also found that occasionally the serum of the mother and less frequently the serum of the father or older siblings was also positive.

The spinal fluid or heparinized blood or both are injected into mice and guinea pigs by the intracerebral and abdominal routes. Using rabbits, neutralization tests can also be made with human sera. For details of these tests, reference should be made to Sabin's article.

Conclusion. Cases of chorioretinitis in infants in which the toxoplasma parasite has been found microscopically have been described. Other cases in older children and adults whose sera contained neutralizing bodies for toxoplasma are mentioned in the literature. This new etiologic concept is important enough to stimulate further study.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

October 21, 1943

DR. ALFRED COWAN, *chairman*

MEMOIR OF DR. GRISCOM

DR. CHARLES R. HEED delivered this tribute.

DRUSEN OF THE OPTIC NERVE SIMULATING BRAIN TUMOR

DR. J. WALDMAN and (by invitation) DR. N. S. SCHLEZINGER and DR. B. J. ALPERS presented a paper in which the anamnestic data in two cases of optic-nerve drusen were described and revealed a clinical syndrome chiefly characterized by headache, vomiting, and an altered appearance of the optic discs somewhat suggestive of early papilledema. The diagnostic possibility of a brain tumor was seriously considered in both cases. The result of intensive clinical investigation, including pneumoencephalography, made evident the fact that the symptoms in these patients were not due to brain tumor.

A review of the literature discloses that the origin of optic-nerve drusen is as yet not clearly defined. The consensus of opinion favors the hypothesis that drusen arise from abnormal hyaline secretions of the proliferated neuroglia in the papilla. The occurrence of optic-nerve drusen without symptoms is well known, and their association with ophthalmologic diseases such as retinitis pigmentosa, glaucoma, melanosarcoma, and so forth, has

been pointed out. The relatively frequent concurrence of drusen of the optic nerve with neurologic disorders is considered in some detail. Particular attention is directed toward the relatively frequent appearance of headache as a conspicuous symptom in some patients with optic-nerve drusen. Such patients demonstrate the fact that this generally benign neuro-ophthalmologic disorder may sometimes be characterized by symptoms and signs which closely resemble those ordinarily associated with brain tumor. Conspicuous among these clinical manifestations are: (1) headache; (2) obscuring of the optic disc with, at times, elevation of the optic disc; and (3) various visual-field alterations, usually including enlargement of the blind spot.

Discussion. Dr. William Zentmayer said that he thought that cases showing the accumulation of hyaline bodies *en masse* on the head of the optic nerve are especially rare, and make it worthwhile reporting such cases. He had two such instances that he wished to put on record. The first patient was a 23-year-old woman who was referred in May, 1942, by an ophthalmologist in Florida. She had been under his care since July, 1928. In 1931 there was an appearance of pseudopapillitis, but it was not until 1939 that the present condition in its incipiency was noted. In April, 1942, the ophthalmologist's notes detailed an accurate description of the condition. The visual fields were contracted and showed a number of reëntering angles. Suspecting some organic trouble, he had a thorough physical examination made which revealed some kidney disturbance, the exact diagnosis of which was not stated, but there was a persistent high albumin with blood cells.

The patient was normal mentally, and presented no physical defects. The corrected vision was R.E. 6/4, partly; L.E. 6/6, partly. The intraocular pressure was a low normal. In the right eye, the media were clear, and on the upper portion of the disc there was a mass of hyaline matter resembling a "white mulberry." The mass lay beneath the vessels, and there was some extension onto the retina. In the left eye, the appearance of the disc was similar, but not so conspicuous as in the right eye. In both eyes the retinal arteries were too bright.

The visual fields showed considerable irregular contraction with great enlargement of the blind spot; the right field presenting several reëntering angles, and the left field but one. There could be but little doubt that the condition was drusen of the nerve head. The visual-field anomalies have been noted before by others, the explanation is that the mass makes pressure on the retinal fibers as they emerge from the disc. As the history seemed to indicate that the condition was recently acquired and progressive, there is a remote possibility that the central vision might later be impaired by pressure on the papillo-macular bundle of fibers.

The second case was, one might say, more spectacular. The patient was a nine-year-old boy, referred by an ophthalmologist in New Jersey. The history was negative. The child was somewhat undernourished, but there was no history of convulsions; his mentality was good, and there was no adenoma sebaceum. In the right eye the media were clear. Covering all but a narrow rim of the optic papilla, and extending somewhat below the lower border of the papilla onto the retina, was a heap of hyaline globular bodies, varying in size, but averaging about the diameter of a retinal vein, the larger ones being at the lower portion of the mass, giving the whole the appearance of a "white mulberry." The spheres were almost transparent, and projected 5 diopters into the vitreous. The fundus level was emmetropic. At the temporal border of the disc,

the crystals were banked in two levels, and, between the two, the inferior temporal vein emerged. The nasal vein was pushed up somewhat at the edge of the disc. The rest of the fundus was normal. After homatropine had been instilled, the vision in the right eye, without correction, was 6/6.

The fundus of the left eye presented no anomalies. No recent notes regarding the progress of these cases were made. Mass drusen of the nerve are rather uncommon, and this is shown by the fact that the men who saw the two cases had many years of experience in ophthalmoscopy, but apparently neither of them had ever seen such a case.

In regard to the first patient, the notes showed a pseudo-optic papillitis. As the patient was under observation for several years before the appearance of the hyaline masses on the disc, it is possible that the pseudopapillitis observed in 1931 was due to the presence of these hyaline masses in the optic nerve, and that later the increase became apparent on the surface of the papilla.

He said he believed there should be no difficulty in making a diagnosis in these cases, although in tuberous sclerosis the gross ophthalmoscopic appearances are similar, in some instances they have been described as "mulberry like," and should there be but a single lesion involving the nerve head, there would be a close resemblance. However, the general symptoms of tuberous sclerosis at once clear up the diagnosis.

Dr. N. S. Schlezinger, in closing, said that he would like to add one thing, that it is the case in which the drusen are more deeply situated, and in which the appearance of the fundus resembles a pseudoneuritis that creates a problem from the neurologist's standpoint. In such cases there may be some elevation of the optic disc and some enlargement of the blind spot, but no drusen visible ophthalmoscopically. He said he thought that these present a difficult diagnostic problem especially if they are associated with the type of headaches that were such a conspicuous complaint in the second case he described.

Massive skin graft

Dr. E. B. Spaeth gave a moving picture demonstration which illustrated some of the rather important factors connected with a free-skin graft. The picture presented a patient with a tremendous keloid in the whole right eye which was in a terrific state of ectropion from keloid and contraction.

An attempt was made subsequently for corneal grafting, but this proved unsuccessful because of the tremendous thickening of the cornea accompanied by a severe iridocyclitis. Insofar as the skin graft was concerned, it was simply a matter of improving the cosmetic appearance of the child.

Junius Kuhnt macular degeneration

Dr. Samuel Altman presented a paper on this subject.

Discussion. Dr. Charles E. G. Shannon stated that he had been permitted to examine the patient and there were two features that impressed him and that were brought out by Dr. Zentmayer in a recent discussion of this case. First, that the macular degeneration should occur in a man 34 years old (the juvenile type—that is extremely rare); and, second, the opportunity of noting the early or hemorrhagic stage in the right eye and its full development in the left.

Dr. W. Zentmayer stated that it must be rare to be able to observe the initial stage and the final stage of this condition in the same patient, so that Dr. Altman's case was a very instructive one.

As to the origin of the fibrous tissue,

it was long held that it arose from the hemorrhages. Later, Verhoeff found that the pigment epithelium of the retina was also a factor in the production of the new-formed tissue.

Dr. Samuel Altman, in closing, said that it seemed that in these cases, once the damage is done, and vision reaches a certain level, the condition remains stationary.

Warren S. Reese,
Secretary.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

October 4, 1943

Dr. Sigmund A. Agatston, *presiding*

Contact lenses

Mr. Theodore Obrig presented a kodachrome film on this subject during the instructional hour.

A new method for testing macular function

Dr. Max Goldschmidt pointed out that in any beginning affection of the macula, confusion may exist between a macular lesion and retrobulbar neuritis. He quoted Duke-Elder as saying "the clinical picture of the retinal edema in its milder aspects is frequently difficult to detect; even with Vogt's red-poor light and Evans's method of scotometry, the finding of a macular edema is difficult or even impossible."

These difficulties may be overcome by applying the phenomenon of Haidinger's brushes for the examination of the macula. This phenomenon is elicited by gazing through a polaroid glass or a Nicol's prism which is rotated before a regular white plane or the blue sky. The eye sees two bright patches, bluish in color, bordered by two hyperbola, between which are the characteristic yellow brushes. The figure rotates with the polaroid glass. To facilitate the observation of the brushes, Dr. Goldschmidt combined the polaroid glass with a blue-glass filter and the patient looks through a tube at a milk-white plane glass illuminated from behind.

The ability to see the Haidinger's brushes is relatively independent of the visual acuity and the state of refraction, but requires the integrity of the macula. Examination of the average patient or older child is not difficult, but where there is involvement of both eyes, or the patient has only one eye, there must be sufficient intelligence to understand the procedure.

The phenomenon is dependent upon the arrangement of the fibers of Henle's layer which parallels the external limiting membrane and acts as a polarizer, the polaroid glass acting as an analyzer. The slightest change, structural or physiologic, results in loss of this faculty. It is therefore possible to apply the Haidinger's brushes in cases where there is questionable lack of the fovea, the slightest pathologic change in the fovea, or for differentiation between the earliest stages of macular disease and retrobulbar neuritis.

The brushes could not be seen in cases of albinism with nystagmus. In alternating strabismus they could be seen with either eye and with the fixating eye in monocular strabismus, but only some of the patients in latter cases saw them with an amblyopic squinting eye. These had improvement of vision on prolonged occlusion, but amblyopic eyes which could not see the brushes did not benefit by occlusion of the other eye. A patient having retinal edema, discernible with the Gullstrand ophthalmoscope but not the monocular ophthalmoscope could not see the brushes. However, they were seen when the edema subsided and were lost again upon recurrence.

The brushes are never absent in retrobulbar neuritis except where there is an

absolute central scotoma and marked visual loss. Their presence or absence proved of value in differentiating between this condition and early macular disease.

Other applications of this test, which Dr. Goldschmidt suggests calling *Haidinger's polarization test of the fovea centralis,* will be reported later. These concern the influence of drugs on the macula, the measurement of intraocular capillary pressure, and the possibility of an early diagnosis of glaucoma.

Discussion. Dr. E. Waldstein pointed out that gazing through a pinhole in a piece of moving cardboard enables one to see the vascularization about the macula. An intelligent patient with one normal eye should be able to observe the difference between it and an eye with a disturbance in the vessels.

Dr. Goldschmidt pointed out that the observation Dr. Waldstein mentioned does not help in examining the macula itself, but rather the endings of the blood vessels.

Contact glasses in diagnosis

Dr. John McLean discussed special uses for contact glasses in diagnosis. The spherical segment ground glass may be used to eliminate corneal astigmatism in the determination of lenticular astigmatism. The same type of glass is sometimes useful in obtaining a clear view of the fundus in keratoconus and other conditions of corneal aberration which distort the ordinary ophthalmoscopic picture. The glass with four lead dots at the limbus is well known for its use in X-ray localization of intraocular foreign bodies. The flat-surfaced contact glass which optically eliminates the anterior corneal curvature renders the fundus and posterior vitreous visible for slitlamp examination. Such technique, however, requires close approximation of the illuminating and visual axes and permits only a monocular picture.

A gonioscopic contact glass is the only absolute necessity in the way of gonioscopic equipment. For ordinary clinical use complicated gonioscopes are not necessary, and a variety of illuminating and magnifying devices can be used. Gonioscopy is not confined to the study of glaucoma. It is also of great clinical value in the study of anomalies of the anterior chamber and its angle; the discovery and localization of small foreign bodies in the angle; the study of the angle in inflammatory processes; tumors of the angle, iris root, and ciliary body; and in postoperative studies after all types of intraocular surgery.

In the study of primary glaucoma it was pointed out that gonioscopy may not be relied on for early diagnosis because the majority of early cases do not show changes in the angle. However, the selection of the best type of operation and the right site for it is best done with the aid of gonioscopy.

In congenital glaucoma the type and extent of congenital anomaly of the filtration structures is disclosed by gonioscopy and treatment may be undertaken accordingly.

In postoperative glaucoma, particularly following cataract extraction, glaucoma may often be suspected before any other signs are manifest by study of the angle. This procedure reveals early changes in patients who must be watched and treated for potential postoperative glaucoma.

Various other types of glaucoma can be discovered and understood, such as, glaucoma capsulare, diabetic rubeosis, and glaucoma following central venous occlusion.

In unsuccessful glaucoma operations gonioscopy is often useful in diagnosing the cause for surgical failure so that it can subsequently be avoided.

Slitlamp study of the vitreous

Dr. Milton Berliner discussed the biomicroscopic examination of the vitre-

ous and the limitations of the ordinary slitlamp and microscope. Methods of overcoming these difficulties to enable visualization of the posterior vitreous were described. The normal vitreous as well as various pathologic changes were considered and slides were presented illustrating these as well as other aspects of the paper.

DIAGNOSTIC VALUE OF PUPILLOGRAPHY

DR. OTTO LOWENSTEIN said that pupillography is a method of simultaneously recording the pupillary movements of both eyes under the condition of darkness by means of an infrared-light filter. It is a diagnostic system based on the results obtained by that method. Routine pupillographic examination includes the study of the reactions to: 1) light; 2) near and distance vision; 3) darkness; 4) sensory and psychologic stimuli; as well as 5) the pupillary reflex to lid closure, either after tactile stimulation of the cornea or the lid margins, or as spontaneous blinking.

It is the purpose of pupillographic examination to check the integrity of the more or less well-known pathways of nervous control of the pupils, on both the parasympathetic and the sympathetic side.

A short, up-to-date review of the results of pupillographic studies was given showing that pupillography is a kind of microscopic analysis of a living process which enables one to detect phenomena invisible to the naked eye. They are concerned with:

1) The early diagnosis and localization of organic lesions of parasympathetic origin. In this group the detection of preliminary stages of the Argyll Robertson pupil seemed to be the most important one, enabling early, really preclinical, detection of symptoms of a syphilitic lesion of the central nervous system and, particularly, observation of its development.

2) The vast field, only recently opened by pupillography in the diagnosis of the sympathetic system, makes it possible to arrive at an early diagnosis within a region hitherto largely unknown and to evaluate many symptoms. This may reveal other conditions, especially neuroses.

Discussion. Dr. Mark Schoenberg summarized the findings from 50 cases of glaucoma studied pupillographically with Dr. Lowenstein. Changes were found in all 50 of these cases of glucoma simplex, not only in established cases but in very early cases, and, even more significant, in the apparently normal eye of patients with clinically unilateral glaucoma. This last finding holds great promise for the future, although it must be realized that it is based on only 50 cases.

Dr. George Kleefeld described studies performed on visualization of the pupil in darkness, using infrared light for observation. These studies were interrupted by the invasion of Holland and await the termination of the war for their resumption.

Dr. Isadore Givner stated that he and Dr. Lowenstein have been investigating Graves's disease pupillographically. The number of cases is small and his remarks were made as a preliminary report. Pupillographic changes are found showing fatigue of the contraction to light, which indicates parasympathetic involvement. In all cases studied, there was a block in the normal redilatation that occurs after contraction to light. This is significant of central sympathetic disturbance. If these findings are confirmed by further work, they will be the first tangible evidence of hypothalamic involvement in Graves's disease and may explain not only cases of widened palpebral aperture with no elevation of the basal metabolic rate, as in postthyroidectomy cases, but also the absence of a dilated pupil in a condition in which stimulation of the sympathetic has long been recognized.

Leon H. Ehrlich,
Secretary.

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THE TRAINING OF BRITISH OPHTHALMOLOGISTS

In view of the coming postwar coöperation between America and Britain, it may be appropriate to study the similarities and differences of ophthalmic education in the two countries. Before taking up the question of ophthalmic residencies, it is necessary to understand the background of general medical education in England.

The prescribed course of medical studies usually covers five years; the 1st year is entirely premedical, the 2d and 3d years are devoted to the regular basic medical studies, and the last two years are occupied with clinical work in a hospital. None of this period is spent as an actual resident, and after graduation it is not compulsory to take an interneship, although the majority do apply for the small number of good interneships available. No A.B. or B.S. degree is required for admission to medical schools, hence matriculation often takes place at about 17 or 18 years of age following the completion of what would correspond to our high school course. Thus it is possible to begin general practice at the age of 22 or 23. The joint degree of M.B. and Chir. B. (Bachelor of Medicine and Surgery) is granted to

those who have completed the required course of study and passed the necessary examinations. The degree of M.D. cannot be taken until three years later and is given to those who submit a satisfactory thesis on some medical topic. There is also a corresponding degree in surgery, M.S. or M.Ch. (Master of Surgery), granted on the same basis. These higher degrees are purely voluntary and most British practitioners do not possess them.

After completing his medical studies, the young doctor is not faced with the prospect of state board examinations such as exist in the United States. A man qualifies for medical practice as soon as he obtains a degree in medicine and surgery from a university. He simply has to register this degree with the General Medicine Council and can then begin practice immediately anywhere in the United Kingdom. Although some medical schools are considered superior to others, there are no third-rate schools such as we understand them; hence the possession of any university degree in medicine and surgery is considered evidence of qualification to treat the sick.

In addition to the regular university medical degrees, there are several medical diplomas, the possession of any one of which entitles a man to registration as a medical practitioner on exactly the same basis as the holder of a university degree. The first of these is called the L.S.A. (Licentiate of the Society of Apothecaries). This is considered the lowest "degree" in British medicine. It can be taken by those who have not quite finished their university course, since the full five years of medical studies are not required before application is made for this examination. If one is granted the L.S.A. one can theoretically be qualified to practice medicine a short time before graduation from medical school. Should he fail in his final university examinations, a student may try for the L.S.A. diploma and, if successful, be able to practice while waiting for another try at his university degree. This however is not the usual custom. It should be pointed out, also, that a practitioner who holds only the L.S.A. diploma is not eligible for election to any good medical society nor to the staff of any first-class hospital.

Another of these special medical diplomas is the so-called Conjoint Board Certificate leading to the joint degrees of M.R.C.S. and L.R.C.P. (Member of the Royal College of Surgeons and Licenciate of the Royal College of Physicians). This diploma is conferred after the candidate has successfully passed a comprehensive examination. Here, again, the qualifying period is slightly shorter than the regular university degree, and the standard of the examinations is generally considered lower, partly because the latter may be taken in three or four separate parts instead of all together as is the case in most university degrees.

There are a large number of students in London medical schools who take these Conjoint Board examinations. They do this in order to become qualified a few months earlier than would be possible if they waited for their regular university degrees. They then become eligible to compete at an earlier date for the relatively small number of good hospital appointments (interneships) which are available at the big teaching hospitals. About half of them go back later to take their regular M.B. Chir.B. degree at the university.

The Scottish Colleges of Physicians and Surgeons in Edinburgh and Glasgow offer another diploma, the L.R.C.P. and L.R.C.S. (Edinburgh) and L.R.F.P. and S (Glasgow), often called the "Scottish Triple." This is more or less similar to the Conjoint Board Certificate.

The aforenamed are those degrees and

diplomas that entitle a man or woman to begin the general practice of medicine anywhere in the British Isles. For those who wish to specialize in some branch of medicine or surgery it is customary, though not necessary, to obtain further qualification. There is no statutory obligation to obtain higher degrees, but appointments to the staff of most good hospitals are open only to those who do. All good staff surgical appointments, including the best appointments in ophthalmology, require candidates to possess the F.R.C.S. (Fellow of the Royal College of Surgeons), which is open to any registered doctor who can pass the severe examinations in anatomy, physiology, pathology, and surgery, both clinical and operative. These examinations are organized and conducted by the Royal College of Surgeons in England and a corresponding body in Edinburgh. For those wishing to specialize in internal medicine there is a stiff examination offered by the Royal College of Physicians of London leading to the degree of M.R.C.P. (Member of the Royal College of Physicians.) To those specializing in obstetrics and gynecology there is offered an additional degree of M.R.C.O.G. (Member of the Royal College of Obstetrics and Gynaecology), awarded after passing an examination and submitting clinical notes on a certain number of cases.

Those members of the Royal College of Physicians and Royal College of Obstetrics and Gynaecology who attain posts of high authority in certain hospitals and universities may be elected without further examination to the title of Fellow of their respective colleges (F.R.C.P., F.R.C.O.G.).

In addition to these more well-known diplomas and degrees there are a series of others awarded by the Royal College of Surgeons and Royal College of Physicians jointly. These include diplomas in public health, radiology, ophthalmology, otolaryngology, child health, and anesthetics. Such diplomas can be obtained only by further examination after qualification. In the majority of cases the candidates must have held certain posts in recognized hospitals where they have practiced their specialty for a specified period of time. These special diplomas are quite similar to our American Specialty Board Certificates.

It can be seen from this brief outline that there exists in Britain a graded system of medical education not unsimilar basically to our own. The first difference that strikes one is the early age at which a doctor may begin to practice. Although this undoubtedly lightens the economic burden, the average American would feel that this advantage must be weighed against a lower standard of education in the liberal arts and a certain lack of maturity on starting out to heal the sick. Perhaps only 10 percent of British medical graduates possess an A.B. degree.

The next notable difference is the absence of the compulsory interneship following graduation from medical school. About 75 percent of graduates do take a house job of six months' to one year's duration but the remaining 25 percent usually become assistants to well-established practitioners and gradually acquire experience during their apprenticeships.

It should be pointed out again that while standards for general practice seem rather low, the possession of a qualifying diploma alone, such as the L.S.A. or the Conjoint Board Certificate, does not open up to the young doctor opportunities for advancement, for even in the smaller rural hospitals preference is given to the holder of a university degree in medicine. Hence the majority of young medical men do obtain their university degrees.

If one is going to specialize, the requirements are relatively high. Special

residencies are available where good training can be obtained. Then examinations are taken for the Specialist diplomas and later for the high degrees of F.R.C.S., M.R.C.P., or M.R.C.O.G. These latter degrees might be compared in some ways to our own F.A.C.S. and F.A.C.P. except that the British degrees are considered more as real degrees and are only granted after passing very severe examinations at which sometimes more than 50 percent fail.

So much for British medical education in general; we now come to a consideration of their ophthalmic residencies. Although there naturally exist individual differences in the training routines of many ophthalmic institutions, we might take the Royal London Ophthalmic Hospital (Moorfields) as a typical example. This institution, established in 1805 as the first special hospital for the treatment of the eyes in the United Kingdom, is known to many American ophthalmologists. At Moorfields there are four residents known, in order of seniority, as the Senior Resident Officer, and the first, second and third House Surgeons. They are appointed for periods of six months in each of these positions upon recommendation of the medical board. Thus, a man who secures an original appointment, may expect to have a period of two years' training.

Original appointments are made on the basis of previous training, letters of recommendation, and personal interview with the surgeons. Although the only stated requirement is that candidates must be registered medical practitioners, actually there are a number of other factors which determine their selection. For instance, those possessing a university degree are considered before those holding a medical diploma such as the L.S.A. or the Conjoint Board Certificate. Also preference is given to those who have completed an interneship in general medicine or surgery. In addition, practically all appointees have either had a shorter period of ophthalmic training elsewhere, or have worked, under supervision, in the out-patient clinic at Moorfields for a year, and taken various courses there before applying for the appointment. All residents live in the hospital and, in addition to room, board, and laundry, receive a rather generous salary which, in terms of U. S. currency at current exchange rates, is as follows: the 3d House Surgeon $400, the 2d House Surgeon $500, and the 1st House Surgeon and Senior Resident Officer $600 each. Thus, at the completion of the 2-year appointment, a man will have received $2,100. In addition to this, the Senior Resident Officer is authorized to accept remuneration from the surgeons for assisting them in private operations in the hospital. Also he may collect small fees for attendance in court cases and for making reports on hospital cases when requested. It becomes apparent that the financial remuneration for a resident at Moorfields far exceeds any figure given to American ophthalmic residents. This is rather surprising considering the relatively lower pay scales which exist throughout all phases of military and civil life in England.

The routine of the residents may be outlined briefly as follows:

The duties of the 3d House Surgeon are entirely in the out-patient department. He is kept busy all morning seeing cases referred to him by members of the surgical staff. When required, he assists at minor operations and administers anesthetics to outpatients. In the aftenoon he continues to see the overflow from the morning clinic and takes care of all accident cases or other emergencies that may present themsevles.

The 2d House Surgeon's duties pertain mainly to in-patients. He has allotted to

his care all public house patients on one floor as well as cases of purulent ophthalmia. He is responsible for keeping the records on these patients, making the daily visit, changing the dressings, and the like. He accompanies the surgeon on his visit and assists him in operations. He may also be called up to assist the Senior Resident Officer or the 1st House Surgeon in surgery, or to administer anesthetics when required.

The 1st House Surgeon takes care of the remainder of the public-ward patients and also the private patients. He makes the visit with the surgeon in charge and assists at operations. It is at this point that he is allowed to do some surgery under supervision.

The Senior Resident officer has general control and supervision of all house patients, both public and private. He is responsible for the work performed by other residents. He attends the surgeons on their visits and assists them in operations. It is during these last six months that the bulk of his operating is done. It has already been mentioned that he also represents the hospital in testifying in court cases and in making out reports for insurance cases.

There is no doubt that a resident of Moorfields is well trained in surgery. The following list of operations performed by a recent graduate during his term of office as Senior Resident Officer and 1st House Surgeon represents the average surgical experience.

Operation	*Cases*
Cataract	100
Trephining	50
Iridectomy	30
Squint	100
Retinal detachment	90
Intraocular foreign body	35
Enucleation and Evisceration	120

The bulk of refraction and perimetry at the hospital is done by paid refractionists. Orthoptics and clinical laboratory work (blood counts, cultures, urinalysis, and so forth) are taken care of by special technicians. Thus a resident is not bothered by the necessity of performing these details but is free to employ his time in studying his patients. This system has much to recommend it provided the resident has a thorough training in refraction and perimetry beforehand and is able to do enough refractions and fields during his residency to learn the art of these all-important tests as well as the theory.

There is no organized system of instruction during the residency itself. The elements of refraction, perimetry, pathology, and the like, must be learned beforehand. As previously stated, practically all residents before appointment have worked in the out-patient department for a year. Here they have availed themselves of the various courses offered in the afternoons by the school of ophthalmology. The faculty of this school consists of the active and consulting staff of the hospital. A list of these courses is as follows:

Anatomy
Optics
Ophthalmoscopy
Medical ophthalmology
Operative surgery
Slitlamp microscopy
Orthoptics
Physiotherapy
Contact-glass clinic
Physiology
Errors of refraction
Pathology lectures
External diseases of the eye
Diseases of the retina and optic nerve
Practical pathology (1 month in lab.)
Practical bacteriology (1 month in lab.)
Motor anomalies, Squint, etc.
Radiography and Radiotherapy
Diseases of the uveal tract

The total fee for all these courses amounts to about $175. The completion of this curriculum requires at least six months, and admits candidates to the examinations for the D.O.M.S. (Diploma in Ophthalmic Medicine and Surgery). These courses, which are practical as well

as didactic, combined with observation of patients in the out-patient department and in the house, give a man a good basic training before beginning his residency.

In comparing the British medical-education system to ours one gets the impression that while the training of the English general practitioner may be inadequate, that of the English ophthalmologist is of high quality, as judged by the standards of Moorfields. We might learn much from our English cousins and they from us. The creation of exchange fellowships between the better teaching hospitals of England and America could not fail to be of mutual benefit to ophthalmology in both countries.

Edwin B. Dunphy.

BOOK NOTICE

MEDICAL PHYSICS. Editor-in-Chief, Otto Glasser, Ph.D. 1744 pages. Chicago, Illinois, The Year Book Publishers, Inc., 1944. Price not stated.

This is a very large volume covering most medical and allied specialties; such as, anatomy, bacteriology, biometrics, biophysics, dermatology, and so forth. The section on ophthalmology was written by Albert D. Ruedemann, M.D., Head of the Department of Ophthalmology, Cleveland Clinic Foundation, Cleveland, Ohio. There are also numerous articles having to do with optics. These are edited by W. B. Rayton, D.Sc. It is obvious that in such a compendium as this there are many subjects not under either of the above headings that would have more or less bearing on ophthalmology; such as, neurology, radiology, bacteriology, and so forth.

Among the contributors to ophthalmic and closely related subjects is Dean B. Judd, Ph.D., who writes on color vision. As one would expect, the physics of the subject is considered in great detail. Colorimetry is discussed, and the theories of Young-Helmholtz, Ladd-Franklin, Hering, and Müller are given in more detail than can be found in any but the most exhaustive treatises. Retinal adaptation is explained qualitatively and quantitatively. Color-vision tests are very thoroughly considered by Elsie Murray, Ph.D.

Light, Vision, and Seeing represents a chapter contributed by Dr. Matthew Luckiesh and the late Dr. Frank K. Moss. Much of this will be familiar to the readers of this Journal, for many of the articles on this subject were published in it.

A chapter on optical illusions contains many interesting figures as well as some history of the subject and theories to explain this by Otto Glasser, Ph.D.

Dan H. Moore, Ph.D., writes on optical methods in electrophoretic and ultracentrifugal analyses.

The application of optics to ophthalmology is the contribution of Dr. Charles Sheard. The consideration of theories is followed by an excellent discussion of bifocal lenses with some suggestions as to factors to be considered in selecting them.

Detailed explanation of fundus photography is Dr. Ferdinand L. P. Koch's contribution to the volume. This includes a description of the Nordenson and the Zeiss retinal cameras, with advice on technique of making the pictures. The physics of vision, especially its photochemistry, is presented by George Wald, Ph.D.

The ophthalmic articles, as would be expected in a book of this character, are of a highly scientific and technical nature and of particular value to any interested in research along these lines.

Lawrence T. Post, M.D.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

1

GENERAL METHODS OF DIAGNOSIS

Bean, L. L. **A simple night-vision test.** U. S. Naval Med. Bull., 1944, v. 43, July, p. 143.

A simple device for the testing of night vision is described. It consists of a box with two compartments. One compartment contains the light source, a 3.8-volt bulb attached to the ship's current. By means of a sliding bar, an aperture between the compartments can be varied in size from 1/64 to 3/8 inch. This small light source illuminates the ground-glass end of the box, and the criterion selected is the ability of the dark-adapted subject to recognize accurately, with the smallest possible light source (in terms of size of aperture), the position of an opaque arrow on the ground glass.

The test provides no quantitative measurement in terms of luminosity, nor does it provide for the detection of malingerers. On shipboard it has a local applicability for elimination of those visually unfit for duty as night lookouts. Benjamin Milder.

Stern, H. J. **A simple method for the early diagnosis of abnormalities of the pupillary reaction.** Brit. Jour. Ophth., 1944, v. 28, June, pp. 275-276.

In the normal eye, if a pin point of light is projected on to the eye with the slitlamp in such a way that the light enters the pupil near the margin of the iris, the pupil contracts. Since no light then reaches the retina, the pupil dilates. Further, when the light reaches the retina, the pupil contracts. The artificial hippus thus begun continues regularly in the normal eye as long as light enters the pupil.

In cases of latent syphilis and early tabes with clinically normal pupillary reaction, the artificial hippus could not be produced. Usually a good contraction followed the first stimulus. This was succeeded by a few irregular, sluggish contractions, after which the pupil became immobile. This fact may become a useful diagnostic aid if subsequent investigations bear out the observations thus made on ten or twelve cases. Edna M. Reynolds.

2

THERAPEUTICS AND OPERATIONS

Dimitry, T. J. **The therapeutic use of choline in ophthalmology.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 1011-1014. (References.)

Heath, Parker. **Chemotherapy in ophthalmology.** Jour. Amer. Med. Assoc., 1944, v. 124, Jan. 15, p. 152.

A general discussion of chemotherapeutic agents begins the article. The author stresses the importance of watching for toxic side actions with any effective drug. Specific suggestions for chemotherapy in some eye diseases are made, as follows: Oral sulfathiazole in usual dosage is given for inclusion blennorrhea, gonorrheal infections of the eye; oral sulfapyridine or sulfadiazine for keratitis and ulcer due to B. pyocyaneus, orbital abscess, thrombosis of the cavernous sinus, postoperative infections, late infections after trephining, enophthalmitis, and for certain cases of acute uveitis. Trachoma responds to sulfanilamide if secondary invaders are present. Hemolytic-staphylococcus conjunctivitis and blepharitis are treated by Staphylococcus toxoid and sulfathiazole ointment. Pneumococcic conjunctivitis is often helped by tyrothricin 0.3 percent drops in conjunction with oral administration of sulfapyridine or sulfadiazine. Tyrothricin irrigations may help dacryocystitis; and also, in connection with thermophore applications, dendritic ulcers.

Robert N. Shaffer.

Just Tiscornia, B., and Moret, R. L. **Radium therapy in ophthalmology.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Sept., p. 543.

After discussing in detail the accidents which the application of radium to the eyes may produce, the authors report 21 cases of tumors of the eye and its appendages treated with radium in the Municipal Institute of Radiology and Physiotherapy of Buenos Aires. From this experience the authors conclude that the tissues and media of the eye do not possess any special radiosensitivity. Early and late complications are mainly due to faulty technique or to treatments being too frequently repeated. If sight is to be preserved, neoplastic lesions of the ocular region, no matter how minimal, require a very rigidly regulated technique, applied in a single and effective treatment. (Illustrations, references.)

Plinio Montalván.

Swan, K. C., and White, N. G. **Choline esters with mydriatic and cycloplegic action.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 933-940. (5 figures, 4 tables, references.)

3

PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Crozier, W. J., and Wolf, E. **Theory and measurement of visual mechanisms. 12. On visual duplexity.** Jour. Gen. Physiology, 1944, v. 27, July 20, p. 513.

Since the advent, in 1866, of the theory of visual duplexity, postulating that the rods are responsible for scotopic vision and the cones for photopic vision, there has been general agreement on the existence of the two morphologic types, which are separable functionally (as evidenced by the duplex performance contours for flicker frequency obtained on visually duplex animals). It is not implied, however, that each phase of such a duplex curve of visual function is necessarily related

to one histologic type of reception; in fact such duplex curves are obtained in some species which possess only one type of photoreceptor cell.

In visually duplex vertebrates, the two groups of neural effects represented by the duplex flicker contours are independently variable (by changing wave length of the stimulus, the area of retina stimulated, and so on); but they do have a common dependence on the quantitative nature of the changes in flicker recognition by the organism. The implication is that the rod and cone groups of neural effects are closely related in a particular manner at the same general central focus.

The authors believe there is no direct correlation between flicker contour and retinal histology. They have examined the flicker response contours of several types to determine (1) whether or not visual duplexity is correlated with retinal duplexity, and (2) whether quantitative performance properties can be correlated with rod and cone categories of visual excitability. Flicker response contours were plotted for Anolis (a 'chameleon" having a cone retina), and Trionyx (a turtle having a duplex retina, mostly cones). The first question was answered affirmatively by finding a simplex curve for Anolis and a duplex curve for Trionyx. In answer to the second question, it was found that no correlations were possible with zoological position on retinal structure; the quantitative characteristics of the performance contours could not be held to be characteristic of properties intrinsic to rods and cones. Thus, in visual duplexity, two groups of neural effects are available, but the curves do not describe properties of the specific photoreceptor units. Benjamin Milder.

Cushman, Beulah. **Aniseikonia.** Med. Woman's Jour., 1944, v. 51, June, p. 17.

The examination for aniseikonia is indicated only in patients with binocular vision, after examination for refractive or muscular defects has been made, and after correction with glasses has failed to bring comfort. Symptoms may be photophobia, headaches, or inability to read for any length of time. Some patients with ocular discomfort and no refractive error may have aniseikonia which can be relieved by glasses that equalize the size difference of the retinal images. Some patients with quite typical ocular migraine have been so relieved. Many referred patients have been found to have a definite size difference in ocular images; 20 percent have been made perfectly comfortable with iseikonic lenses; 60 percent have obtained partial relief and 20 percent no relief. The history is given of a patient aged 17 years, affected by discomfort for continued near work although a defect of refraction and a vertical-muscle imbalance had been corrected. Examination for aniseikonia and inclusion of the corresponding correction in his lenses resulted in complete satisfaction.

Melchiore Lombardo.

Evans, J. N. **Medical aspects of refractive problems.** Sight-Saving Review, 1943, Fall-Winter, v. 13, p. 147.

The time when the "eye man" fitted into the medical picture only through his ability to diagnose kidney disease and blurred discs in addition to his usual fitting of glasses has passed. Although the effect of specialization is narrowing no specialty has a right to exist unless it keeps its field intimately related to medicine as a whole.

To illustrate, the author takes the case of a patient who finds he is able to read without the help of his previous

"far-sighted" glasses. To be ruled out are glaucoma, early cataract, diabetes, inflammatory conditions in the eye, media changes due to medication concurrent or previous, and so on. In examination for "spots before the eyes" one must consider inflammations and hemorrhages, which bring up the whole field of the blood dyscrasias, hypertension, and arteriosclerosis. "Tired eyes" lead one to the anemias or possible cerebral lesions. "Double vision" suggests elaborate study as to neurologic disorders, from cerebral vascular accidents and tumors to multiple sclerosis. Owen C. Dickson.

Gillett, M. M. **Seeing defects in non-readers.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 1007-1010.

Guibor, George. **Phenomena associated with eccentric fixation.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 986-991. (13 photographs.)

Lebensohn, J. E., and Sullivan, R. R. **Temporary stimulation of emmetropic visual acuity.** U. S. Naval Med. Bull., 1944, v. 43, July, p. 90.

The authors observe that all the methods cited as being of value in improving visual acuity have one factor in common, namely visuopsychic excitation. Sympathomimetic drugs and cortical stimulants should, therefore, also stimulate subjective visual responses.

Benzedrine and desoxyephedrine have been shown to produce an increase in the fusion frequency of flicker, a fundamental measurement of visual function. Alcohol may produce a decrease in visual acuity. Analeptics, such as coramine, function by virtue of their stimulation of the higher centers. The authors studied benzedrine and coramine as to their efficacy in improving visual acuity and speed of vision. The former was measured on the Ferree-Rand chart of double broken circles; the latter was tested on the Lebensohn unlearnable letter chart, using a stop watch. The results indicated consistent improvement in acuity and reading speed, the improvement lasting over six hours for each drug. The authors suggest that such an improvement in absolute acuity in emmetropes may be advantageous in periods of exceptional stress.

Benjamin Milder.

Linksz, A., and Triller, W. **"Biastigmatism." Evaluation and criticism of the refractive technique advocated by Márquez.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 992-1002. (2 tables, references.)

Sloan, L. L. **A quantitative test for measuring degree of red-green color deficiency.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 941-947. (5 tables.)

Swan, K. C., and White, N. G. **Choline esters with mydriatic and cycloplegic action.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 933-940. (5 figures, 4 tables, references.)

4

OCULAR MOVEMENTS

Davis, W. T. **Amblyopia ex anopsia.** Ophth. Ibero Amer., 1944, v. 5, no. 4, pp. 327-339 (in Portuguese); pp. 339-344 (in English).

The late author recognized two types of amblyopia ex anopsia, one resulting from interference with the vision of one eye during the period of visual development, the other due to organic changes affecting central vision. Emphasizing

the mental effect of neglected strabismus upon the child, he discusses the usual routine of refractive correction followed by surgery or orthoptics or both according to necessity. He calls "dissociated hyperphoria" that which is the result of a lateral strabismus and not its cause; this type of hyperphoria should never be subjected to operation on the vertical muscle. (References.) W. H. Crisp.

Ellett, E. C., Rychener, R. O. and Robinson, J. S. **Appraisal of value of an orthoptic clinic in private practice.** Arch. of Ophth., 1944, v. 31, June, pp. 503-508; also Trans. Amer. Acad. Ophth. and Otolaryng., 1944, May-June, p. 299.

An orthoptic clinic, available to all ophthalmologists in the city, has been operating in Memphis, Tennessee, for the past ten years. The clinic is conducted by two trained technicians and, if space and talent were available, the number of attendants could well be increased. The instruments used are briefly described. Treatment of anomalous retinal correspondence, of divergence excess, of divergence insufficiency, and of true and latent convergence insufficiency is discussed. The authors state that the Memphis Orthoptic Clinic has been a valuable adjunct in the handling of their private patients. (2 illustrations, references.)

John C. Long.

Magnus, John A. **Congenital paralysis of both external recti treated by transplantation of eye muscles.** Brit. Jour. Ophth., 1944, v. 28, May, pp. 241-245.

A case of bilateral abducens paralysis in a child aged 12 years is reported. A transplantation of the outer halves of the superior and inferior rectus muscles to the insertion of the paralyzed external rectus was done in the left eye and at the same time the external rectus was advanced and recession of the left internal rectus was done.

Transplantation was later performed on the right eye, combined with myectomy of the right inferior oblique. Because the left eye showed a slight hyperphoria, myectomy of the left inferior oblique and partial tenotomy of the left superior rectus were done later. The end result was satisfactory from a cosmetic point of view but no abduction was possible. Because of suppression of the image of the right eye, the patient had no double vision. (18 photographs, 2 drawings.)

Edna M. Reynolds.

Rosenbaum, H. D. **Strabismus in the Army.** Military Surg., 1944, v. 95, July, pp. 48-52.

A recession operation for convergent strabismus was performed on 21 soldiers, of whom 20 required recession and resection with advancement of the weaker opponent. On the basis of the results obtained five of the men were reclassified to general service and two have gone to officer's candidate school. (References, 4 figures.)

M. Lombardo.

Wald, G., and Burian, H. M. **The dissociation of form vision and light perception in strabismic amblyopia.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 950-963. (3 figures, 1 table, references.)

White, J. W. **Clinical application of the screen (cover) test described in detail.** Amer. Jour. Ophth., 1944, v. 27, Sept., pp. 977-986.

5

CONJUNCTIVA

Aparisi, Tomás. **Contributions to the treatment of vernal conjunctivitis.** Arch. de la Soc. Oft. Hisp.-Amer., 1943, v. 3, Aug-Sept., pp. 190-193.

Basing the treatment of vernal conjunctivitis on the morphologic analogy between this condition and trachoma, the author used sulfanilamide in the same doses as used for trachoma. Marked improvement in symptoms is claimed, but recurrences were not prevented. (References.)

J. Wesley McKinney.

Aprigliano, Orlando. **Bisulphate of quinine in local treatment of trachoma.** Arquivos Brasileiros de Oft., 1944, v. 7, April, pp. 45-60.

The author describes in detail the routine treatment of trachoma with this drug in the Trachoma Dispensary of the Health Center of Jaú, State of São Paulo, Brazil. The treatment was introduced in 1935 by Elias Selinger of Chicago. Special nurses are trained to carry on mass treatment of large numbers of patients. The drug is used in solutions of 5 or 7.5 percent, and is massaged into the palpebral conjunctiva and the caruncle with saturated cotton swabs. Most of the Portuguese patients tolerate the treatment without a local anesthetic. As with other drugs used for trachoma, care must be taken to spare the cornea. The author summarizes the clinical course of thirty cases. (Bibliography.) W. H. Crisp.

Bauer, C. E., Gall, A. E. and Cox, C. D. **Meningococcus conjunctivitis.** Military Surg., 1944, v. 95, July, pp. 24-27.

The right eye of an infantry private of 38 years had been painful for three days, with pain increasing in severity during the previous 24 hours. The eyelids were swollen and the conjunctiva moderately chemotic and congested, showing a membrane-like purulent exudate in the lower cul-de-sac. The corneal epithelium was edematous, with punctate areas of greyish infiltration. V.O.D. was 20/200, O.S. 20/30. A smear from the right eye showed Gram-negative intracellular diplococci, Neisserian in configuration. Cultures gave a growth which showed agglutination in polyvalent antimeningococcus serum. Warm irrigations, instillations of argyrol, sulfathiazol orally, and 25 millions of killed typhoid bacilli intravenously, stopped the growth of the organisms after 24 hours and the formation of exudate in 72 hours. Two other cases are reported with very similar history and bacterial findings. Some cases of conjunctivitis have been improperly termed gonorrheal on the basis of tinctorial and morphologic characteristics. The writers believe that meningococcic conjunctivitis is more common than heretofore considered.

M. Lombardo.

Blumberg, M. L., and Gleich, M. **The simplified treatment of gonococcic ophthalmia neonatorum with chemotherapy.** Jour. Amer. Med. Assoc., 1943, v. 123, Sept. 18, p. 132.

A comparative study of three groups of cases of gonococcic ophthalmia neonatorum is given to show simplification and efficiency of the new therapy of the disease. The first group of 14 cases was treated by frequent irrigation with boric-acid solution and 10 to 25 percent mild protein silver. The unaffected eye was protected with some such device as a Buller shield. The duration of the illness ranged from 6 to 46 days. In 35 percent of the

cases complications developed such as arthritis, metastatic abscess, involvement of the second eye, and corneal ulcer. The second group of 14 cases was treated with various sulfonamides orally, in doses of 1 to 1.5 grains per pound of body weight. All were treated with frequent irrigations of boric-acid solution and some with 10 to 20 percent mild protein silver. Only one baby developed a complication, namely involvement of the second eye on the second day of treatment, but both eyes cleared uneventfully. The duration of the disease ranged from one to seven days. The third group of ten cases was treated uniformly, regardless of the infant's weight and severity of symptoms, with 3 grains of sulfathiazole followed by 1 grain every four hours until the symptoms disappeared and smears from the conjunctiva were negative. The eyes were not irrigated and the unaffected eye was not protected. The duration of the disease was one to four days and no complications were observed. No recurrences were noted. The authors feel that mechanical protection and local therapy are unnecessary. Robert N. Shaffer.

Braley, A. E., and Sanders, M. **Treatment of epidemic kerato-conjunctivitis.** Jour. Amer. Med. Assoc., 1943, v. 121, March 27, p. 999.

The authors suggest the use of convalescent serum as a possible means of treating epidemic keratoconjunctivitis. Because no other local or general therapy has been effective, the authors are giving this preliminary report based on results in ten cases. All cases had typical lid edema and bulbar chemosis with preauricular lymph node involvement. There was severe ocular discomfort. Conjunctival scrapings showed only mononuclear cells, and cultures were negative. Corneal involvement to greater or less extent was present in six out of the ten cases. Treatment consisted of intravenous injection of 15 to 50 c.c. of convalescent serum with a titer of 10,000, though in two cases a titer of only 1,000 was used. Improvement began shortly afterward. Seven patients were clinically cured; the normal course of the disease was shortened in two; one case is classed as a failure.

Robert N. Shaffer.

Calkins, H. E., and Bond, G. C. **Adaptation of virus of epidemic keratoconjunctivitis to development in extra-embryonic fluids of chick embryo.** Proc. Soc. Exper. Biol. and Med., 1944, v. 56, May, p. 46.

A strain of epidemic-keratoconjunctivitis virus was isolated by tissue culture and was given two mousebrain passages. A 10-percent suspension of a whole macerated brain was inoculated into the chorio-allantoic fluid of eight-day chick embryos. The embryos died in five to six days. The chorio-allantoic amniotic fluids were collected and pooled and twice again inoculated in chorio-allantoic fluid, then were passed through three mouse-brains and maintained in twelve consecutive embryo-fluid passages. This procedure increased the virulence, so that the present material is lethal for chick embryos in 0.001 c.c. doses, killing them in three to four days.

Human convalescent and hyperimmune rabbit serum will neutralize the embryo-fluid passage strain. The ease of handling this virus in extra-embryonic fluids and the success of neutralization tests suggest that this method might be of value in serologic diagnostic work. R. Grunfeld.

Fritz, W. H. **Epidemic keratoconjunctivitis.** Med. Bull. Veterans Admin., 1944, v. 21, July, p. 48.

The author states that epidemic keratoconjunctivitis is not new, having been described in Austria in 1899. The symptoms, signs, and course of the disease are reviewed briefly, stressing glassy edema of the conjunctiva, predominantly unilateral occurrence, and frequent regional lymphadenopathy. The corneal lesions occur about eight days after onset of the conjunctivitis, and usually do not stain; they persist three to four months. The incubation period is five to twelve days, the cause probably virus. The disorder must be differentiated from acute catarrhal conjunctivitis and Beal's follicular conjunctivitis, neither of which shows corneal involvement. Symptomatic treatment has included atropine, quinine ointment, irradiation, and sulfa drugs. Recently cures have been reported with intravenous convalescent serum. Benjamin Milder.

Toledo, S. de A. **Treatment of trachoma with sulfanilamide.** Arquivos Brasileiros de Oft., 1944, v. 7, April, pp. 68-74.

This article contains the instructions given to eye physicians in the Trachoma Section of the Department of Health of the State of São Paulo, Brazil.

Valentine, F. C. O., and Edwards, A. M. **Angular conjunctivitis treated with propamidine.** The Lancet, 1944, v. 247, June 10, p. 753.

The authors report the successful treatment of ten patients with angular conjunctivitis by the use of propamidine isethionate (4:4′-diamidinodiphenoxypropane di-(Beta-hydroxyethanesulfonate).

One drop of 0.15 percent solution was instilled, after irrigating the eyes three times a day with boric-acid solution. Cultures were negative for Morax-Axenfeld bacillus in two to six days. There were no ill effects from the treatment, but in no case was it used for longer than 14 days. It is felt that the drug may prove useful in the treatment of other types of bacterial conjunctivitis. Benjamin Milder.

6

CORNEA AND SCLERA

Castroviejo, Ramón. **Keratectomies for treatment of corneal opacities.** Arch. of Ophth., 1944, v. 32, July, pp. 11-21.

After a résumé of the literature, the author presents in considerable detail, by description and by serial photographs, his technique in various types of corneal opacities. He discusses partial superficial keratectomy for treatment of band keratitis, dystrophia adiposa, and leukoma. Castroviejo also gives his technique of total superficial keratectomy for vascularized leukoma and its combination with corneoconjunctivoplasty for symblepharon. There is also a discussion of graft of buccal mucous membrane for vascularized leukoma, symblepharon, and recurrent pterygium. Penicillin ointment has been found to shorten the period of healing and to reduce the occurrence of infection. (References and 6 serial photographs.)

R. W. Danielson.

Fritz, W. H. **Epidemic keratoconjunctivitis.** Med. Bull. Vet. Admin., 1944, v. 21, July, p. 48. (See Section 5, Conjunctiva.)

McCreary, J. F., Nicholls, J. V. V., and Tisdall, F. F. **Further studies on**

the relationship of corneal vascularization to riboflavin deficiency. Canadian Med. Assoc. Jour., 1944, v. 51, Aug., p. 106.

The authors have previously reported on the effects of riboflavin on corneal vascularization and symptoms of asthenopia; patients on a riboflavin deficient diet being selected. A marked improvement in both features was noted in those subjects who were given supplementary riboflavin for two months. The corneal vascular changes were recorded by means of a special ophthalmic camera.

In the present study, a contrasting group of patients was selected, in that this group had been on an adequate riboflavin intake (2.9 mg. daily) for a year prior to the investigation. The present study was furthermore carried out by slitlamp examination as well as photographic record. The photographs show the blood-filled limbal vessels well, but the slitlamp was found to be better for the collapsed vessels.

One group of patients was given supplemental riboflavin (9.9 mg. daily) for two months; another group received a placebo. Slitlamp and photographic studies were made without regard to previous data on the subject. The authors found no significant difference in the extent of limbal vascularization in the two groups; and the slitlamp and photographic findings were closely parallel. It is concluded that uniform peripheral corneal vascularization is not a safe basis for a diagnosis of current riboflavin deficiency, although it may indicate a preexisting deficiency.

Benjamin Milder.

Vázquez Barrière, A. **The cornea in acquired syphilis.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Nov., p. 663.

Interstitial keratitis in acquired syphilis usually shows an atypical form; it is generally unilateral and the corneal infiltration is usually partial. In this group is included the syphilitic keratitis punctata of Mauthner, of which the author reports a case with involvement of one eye and later of the other. The initial lesion, a phagedenic chancre, had not yet healed at the time the eyes became involved. Antiluetic treatment cleared up the condition in a short time, with good visual results. The author reports two cases of keratitis pustuliformis profunda of Fuchs. In one case the condition was bilateral and one eye showed a perforation of the cornea with formation of an adherent leucoma; while the lesions in the fellow eye underwent almost complete resolution with antiluetic treatment, useful vision being preserved. In the second case the disease was unilateral, but a secondary glaucoma developed, which required several paracenteses before arsenic-mercury treatment cleared up the lesions. The rapid response of keratitis pustuliformis to antiluetic treatment is stressed. Gummata of the cornea are extremely rare and very difficult to differentiate from keratitis pustuliformis profunda. The author believes that while the subject has not been exhaustively studied, syphilis may produce superficial ulcerative keratitis, in proof of which he mentions the many instances of atonic corneal ulcer which heal only after antiluetic treatment is resorted to. Likewise due to syphilis is a peculiar type of nodular or phlyctenular keratitis of which the author reports a case.

Plinio Montalván.

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Damel, C. S., and Durando, S. A. **Traumatic cysts of the iris and eye-**

lashes in the anterior chamber. Arch. de Oft. de Buenos Aires, 1942, v. 17, Oct., p. 614. (See Section 16, Injuries.)

Hague, E. B. **Uveitis, dysacousia, alopecia, poliosis, and vitiligo.** Arch. of Ophth., 1944, v. 31, June, pp. 520-538.

Because of its completeness and complexity, this monograph is difficult to abstract. A detailed general description is followed by special discussion of each of the various signs in the syndrome.

Hague says the ensemble of symptoms is too consistent to suggest a heterogenous pathogenesis. It is significant that all, or practically all, of the manifestations of the syndrome have followed (a) an infection of the upper respiratory tract combined with suggestion of encephalitis, (b) severe emotional trauma, or (c) a tumor pressing on the hypothalamus. Embryologically, the retina is closely connected with the hypothalamus and the third ventricle.

The author reports a typical case in detail and says that he has tried to account for the bizarre and striking manifestations in terms of a discrete lesion. Some of the bases for the belief are: (1) clinical evidence of oculocutaneous pigmentosis, pointing to early involvement of the hypothalamus; (2) the close relation of the posterior segment of the eyeball, structurally, embryologically, and phylogenetically, to the hypothalamus, and of the latter to centers and tracts controlling or affecting hearing and pigmentation of the eyes, hair, and skin; and (3) functional considerations pointing to retinohypothalamic relations.

Hypothalamic involvement would account for the lethargy and cryesthesia which some of these patients experience. Only one lesion, and that in the suprachiasmic region, can account for all the symptoms of this syndrome.

The frequency with which cases of this syndrome are immediately preceded by an influenza-like episode of encephalitis has been noted. That influenza and certain forms of encephalitis are due to virus infection is now common knowledge. Workers in the field of virus disease contend that certain neurotropic viruses may pass along the nerve pathways connected with the hypothalamus and that they tend to localize in the latter region and there to produce local necrosis. (Bibliography, 3 photographs, 1 figure.)

R. W. Danielson.

Kamellin, Samuel. **Uveitis associated with Hodgkin's disease.** Arch. of Ophth., 1944, v. 31, June, pp. 517-519.

Kamellin gives the differential diagnosis of Hodgkin's disease from glandular tuberculosis, lymphosarcoma, and Boeck's sarcoid. He then presents the case of a 26-year-old man who had both Hodgkin's disease and bilateral uveitis. Whether the uveitis in this case was due to the Hodgkin's disease or was a concomitant disease was not clear. Since, however, the tests and examinations did not reveal any positive indication of other disease and there was pathologic and roentgenographic evidence of Hodgkin's disease, Kamellin is inclined to believe that the ocular condition was due to the Hodgkin's disease. (3 figures, one in color, one reference.) R. W. Danielson.

8

GLAUCOMA AND OCULAR TENSION

Chandler, P. A. **Treatment of glaucoma.** Arch. of Ophth., 1944, v. 32, July, pp. 23-32.

This paper is so substantial that an abstract is inadequate; a few highlights only can be given.

In acute glaucoma, the author uses the Cogan method of instilling glycerine to clear the edematous cornea in order to permit study of the back of the cornea, the aqueous, and the iris with the slitlamp; and also, if the lens is reasonably clear, the fundus. This enables him to make a differentiation between primary and secondary glaucoma. He always attempts to lower the tension with a miotic before operating. Five percent neostigmine bromide combined with 20 percent mecholyl chloride is probably the most effective miotic. One may use 4 percent pilocarpine nitrate or 1 percent physostigmine salicylate.

The rules for surgery in acute glaucoma are as follows: (1) If the attack is less than 48 hours old, iridectomy is chosen whether or not the tension drops to normal under preoperative miotics. (2) If the attack is 48 hours old or more, iridectomy is done if the tension comes fully to normal preoperatively and can be maintained there with reasonable use of miotics. (3) If the attack is 48 hours old or more and the tension cannot be brought to normal with miotics, a filtering operation is chosen. In these conditions it has been the author's experience that iridectomy only succeeds in changing the condition from acute to chronic glaucoma. Trephining has not been successful. The operation most successful in his hands for this type is sclerectomy combined with iridencleisis.

As for surgery, the author stresses the well-known fact that, as a rule, eyes with early glaucoma do much better after surgical intervention than those in which the disease is advanced. Chandler favors trephining for younger and iridencleisis for older patients. When an iris-inclusion operation is performed on a younger patient, sclerectomy is done. Gentleness in handling the iris is stressed.

In chronic glaucoma coexisting with immature cataract, miotics should be used as long as possible before resorting to surgery. If the vision is cut down to 20/50 or less by the opacities in the lens, intracapsular extraction with a broad basal iridectomy will often control the glaucoma. If central vision is 20/40 or better without miotics and miotics reduce the vision below 20/40, broad iridectomy followed by miotic therapy is preferred to immediate extraction of the cataract.

For treatment of buphthalmos or infantile glaucoma, iridencleisis has proved more successful than trephining. In any operation for glaucoma of this type it is regarded as of the greatest importance that adequate massage be carried out by the parents after the child leaves the hospital. In treatment of glaucoma due to iris bombé, Chandler favors iridectomy rather than transfixion of the iris. Glaucoma following discission of the intact lens or following traumatic cataract calls for prompt linear extraction of the lens. In hypermature cataract, combined intracapsular extraction at one operation is favored, rather than preliminary iridectomy. (Fields.)

R. W. Danielson.

Fortín, E. P. **Considerations concerning glaucomatous diseases.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Oct., p. 641.

The author presents several histologic sections of eyes on the basis of which various aspects of glaucoma are discussed. The article does not lend itself to abstracting. (Photomicrographs.) Plinio Montalván.

Igersheimer, Joseph. **Intraocular pressure and its relation to retinal extravasation.** Arch. of Ophth., 1944, v. 32, July, pp. 50-55. (See Section 10, Retina and vitreous.)

Uribe Troncoso, Manuel. **Development of anterior peripheral synechiae in experimental acute glaucoma.** Arch. of Ophth., 1944, v. 31, June, pp. 481-502.

Acute glaucoma was produced experimentally in rabbits, dogs, and monkeys by injections of serum, of defibrinated blood, and of whole blood into the anterior chamber. It was shown that the first step in the production of glaucoma by these agents was not mechanical obstruction of the channels of outflow by the base of the ciliary body and the root of the iris, but a disturbance in the normal outflow of aqueous. This was accompanied by a tissue response of vasodilatation and edema in the base of the ciliary body, which obliterated the ciliioscleral sinus, with resulting adhesions and swelling of the processes. The root of the iris was pushed forward by the thickened processes, making the anterior chamber shallow and producing anterior peripheral synechiae.

Injection of serum into the aqueous, because of the colloidal state thus induced, caused inversion of the normal current, the colloidal aqueous sucking fluid from the intrascleral vessels. The filling of the sinus with serum or blood, and later with fibrin, prevented the normal aqueous in the chamber from reaching the trabecular vessels on the surface of the sclera. This obstruction hindered the outflow of fluid and immediately promoted a great increase in intraocular pressure. The neurovascular response to the experimental glaucoma was different with the different agents employed. Serum alone produced an especially conspicuous response in the iris, while with defibrinated blood the response was greater in the processes and base of the ciliary body. Pure blood coagulated in the anterior chamber and the sinus, occluding the latter and coating the walls of the anterior chamber.

The article contains an anatomical description of the chamber angle in the animals used, details of the technique of injection, detailed reports of the experimental findings, and photomicrographs of the sectioned experimental eyes. (29 figures, bibliography.)

John C. Long.

9

CRYSTALLINE LENS

Meroni, J. C. A. **A case of bilateral cataract following electric shock.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Oct., p. 633. (See Section 16, Injuries.)

Pahwa, M. D. **Intracapsular extraction of senile cataract.** Arch. of Ophth., 1944, v. 32, July, pp. 48-49.

The author has performed 170,000 cataract operations, most of them intracapsular. This paper describes his technique. Anesthesia is by instillation of 2-percent butacaine sulphate, with no retrobulbar or subconjunctival injection and no akinesia. A full 180-degree section is used, the knife coming out 1 mm. below the upper border of the cornea. Iridectomy is performed in all cases. At this stage the speculum is removed, and the assistant lifts the upper eyelid with a strabismus hook held in his right hand and pulls the lower lid down with the fingers of his left hand. The surgeon places his left thumb just above the eyebrow and slightly pulls it up, and in the other

hand he holds the other strabismus hook. This hook is now applied to the sclera 2 mm. below the junction, at about the 6-o'clock position. It is slowly drawn up toward the limbus with very slight backward pressure, which movement dislocates the lens. The lens is then delivered by "tumbling."

R. W. Danielson.

10

RETINA AND VITREOUS

Givner, I., and Roizin, L. **Juvenile amaurotic familial idiocy.** Arch. of Ophth., 1944, v. 32, July, pp. 39-47.

Cerebromacular degeneration, or the juvenile form of amaurotic familial idiocy, was first described by Batten in 1903. The clinical features of this disease were progressive loss of the intellectual faculties, loss of vision, and loss of motor power. Two cases are reported and the histopathologic literature is covered in considerable detail.

From an etiopathogenic point of view, Sachs's concept that the disease is a disturbance of the nervous system of primary and degenerative character still stands true even if viewed in the light furnished by recent investigations. Schaffer's hypothesis that it is a heredodegenerative disease restricted only to neuroectodermal derivatives and transmitted through the hyaloplasm does not seem to Givner and Roizin to be supported by their observations. These authors conclude that the histochemical reactions evidenced by their investigation point to the presence, in involved cells of both retina and brain, of the same type of lipids, that is, a mixture of phosphatides and cerebrosides. (References, 10 photomicrographs.)

R. W. Danielson.

Igersheimer, Joseph. **Intraocular pressure and its relation to retinal extravasation.** Arch. of Ophth., 1944, v. 32, July, pp. 50-55.

The author's interest in this subject was initiated by the study of a middle-aged person with glomerulonephritis who had the combination of high blood pressure, an unusually large amount of nonprotein nitrogen in the blood, primary glaucoma, and absence of retinopathy. From the study of this case and others in the literature, Igersheimer concludes that it appears that high intraocular tension influences the extravasation into the retina and that retinopathy, such as albuminuric or diabetic retinitis, diminishes or disappears when glaucoma develops or does not occur in an already glaucomatous eye.

Another condition undoubtedly related to lowering of the intraocular pressure must be discussed. This is the papilledema which occurs sometimes in eyes with very low pressure (after penetrating wounds or in disease) and which disappears when the intraocular tension becomes normal. Parker has demonstrated that when both eyes have equal tension, papilledema develops to an equal degree in the two eyes. If the tension in one is lower than that in the other eye, papilledema generally develops first in the eye with the lower tension.

The author states that while the condition of the capillaries, the content of the blood, and the resistance to the outflow of blood and lymph from the globe are the main factors whose pathologic alteration may produce increased passage of fluids and corpuscles through the vessel walls of the retina, intraocular pressure also seems to play a certain role in these transudative phenomena. (References.)

R. W. Danielson.

Lepard, C. W. **Lipemia retinalis in the nondiabetic patient.** Arch. of Ophth., 1944, v. 32, July, pp. 37-38; also Trans. Amer. Ophth. Soc., 1943, v. 41, p. 536.

It has been supposed that lipemia retinalis is always associated with diabetes but such is not the case. The level of blood lipids at which lipemia retinalis is first recognized and the level at which it disappears have been carefully determined by several authors. It appears approximately when blood fats increase to above 3.5 percent and disappears when these fats decrease to below 2.5 percent.

In a six-year-old boy, lipemia retinalis was present during the entire time the patient was under observation. On a low fat diet it could be recognized only in the smaller divisions of the central artery and vein in the more peripheral portions of the fundus of each eye. With a high fat diet the arteries and veins throughout their entire extent were increased to twice their size and approached a common creamy color, so as to be indistinguishable from each other. Although lipemia retinalis is usually seen in the young person with severe diabetes, it is a phenomenon resulting from a rise in the level of the blood lipids and may occur in the presence of normal carbohydrate metabolism. Blood lipids containing a high percentage of neutral fat seem more likely to produce characteristic retinal changes than do those containing a low percentage of neutral fat. (References, 3 tables, 1 photograph.)

R. W. Danielson.

Lijó Pavía, J. **Detachment, reattachment, tears, and cyst of the retina.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Nov., p. 708. (See Amer. Jour. Ophth., 1943, v. 26, p. 644.)

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Esposito, A. C. **Neurosyphilis and the oculist.** Med. Bull. Veterans Admin., 1944, v. 21, July, pp. 51-54.

At the Veterans Administration Facility, Chillicothe, Ohio, all neurosyphilitic patients are seen by the oculist before treatment is instituted. A complete examination is recorded, the visual fields being by confrontation or perimeter, depending on the coöperation of the patient.

During a first series of tryparsamide injections, the visual fields, fundi, and central vision are examined after each injection; periodic re-examinations are further made during all subsequent courses of tryparsamide therapy. The author found that 26 of 204 patients on tryparsamide developed toxic involvement of the optic nerve; of this number, only 11 were detected during the first course of therapy. Most of these were found between the third and seventh injections of any course. The resulting atrophy is best treated by malarial fever, as well as continued trivalent arsenic and bismuth therapy. Vitamins and subdural therapy have not proved successful.

Benjamin Milder.

Glicklich, E. A., Schultz, A., and Benjamin, J. E. **Tuberous sclerosis associated with tumor of the optic disc (phacoma).** Arch. of Ophth., 1944, v. 32, July, pp. 60-62.

The authors present a detailed report of a case of tuberous sclerosis in a 22-year-old soldier with the characteristic syndrome of adenoma sebaceum, epilepsy, and mental deficiency, associated with tumor of the optic disc. Though phacomas occur less frequent-

ly in tuberous sclerosis than the cutaneous lesions or the mental disturbances, changes in the fundus may sometimes be the sole manifestation of this syndrome. Under such circumstances tuberous sclerosis must be differentiated from the allied conditions in which retinal phacomas occur, namely, von Recklinghausen's neurofibromatosis, Hippel's retinal and Lindau's cerebral angiomatosis, the Sturge-Weber syndrome (angiomatosis cerebri), and retinal glioma.

The origin of tumors of this type is still not certain, though they are generally recognized as primarily ectodermal malformations, consisting of glial tissue and undifferentiated elements, including mesoderm, initiated by developmental and hereditary factors. (References, 2 illustrations.)

R. W. Danielson.

Prado, D. L. **Arsenical compounds and toxicity for the optic nerve. Contraindications.** Arquivos Brasileiros de Oft., 1944, v. 7, April, pp. 61-68.

The author reviews the various arsenicals in use in Brazil. Trivalent arsenicals (sulfarsenol is an example) have little toxicity for the optic nerve, but should never be used when there are lesions of the ocular fundus. Two illustrative cases are briefly reported, one with fundus photograph. The pentavalents have a more accentuated neurotropism than the trivalents. In the clinic at Santos, State of São Paulo, Brazil, two cases of optic-nerve atrophy in patients subjected to treatment with acetylarsan have been observed. Among 380 patients subjected to stovarsol, only 18 cases of ocular disturbance have been seen, none of which went on to blindness. The author discusses the different theories as to the pathogenesis of disturbances of the optic nerve under arsenical treatment for syphilis.

W. H. Crisp.

Rucker, C. W. **Defects in visual fields produced by hyaline bodies in the optic discs.** Arch. of Ophth., 1944, v. 32, July, pp. 56-59.

The presence of hyaline bodies in the optic disc occasionally produces an appearance that may simulate chronic papilledema with secondary optic atrophy. When the glistening bodies are large and lie near the surface, they cause little difficulty in diagnosis, but when they are small and lie buried so deeply within the nerve substance as to be scarcely visible they may cause a fulness that is difficult to explain. Unless looked for specifically they may pass unnoticed. Not only is the appearance of the optic discs misleading, but field defects are encountered rather frequently in this condition, either in the form of peculiar contractions or of arcuate scotomas. Such defects may confuse the diagnostician still further. In this article, the author gives five typical case histories along with the field defects and fundus pictures. (One reference, 5 figures.)

R. W. Danielson.

Schlezinger, N. S., Waldman, J. and Alpers, B. J. **Drusen of the optic nerve simulating cerebral tumor.** Arch. of Ophth., 1944, v. 31, June, pp. 509-516.

Two cases of drusen of the optic nerve were observed that presented symptoms very suggestive of cerebral tumor. The first patient was a woman of 23 years who had had right hemicrania for years and had suffered from left-sided headaches for three years. The fundi showed bilaterally many lemon-yellow drusen on and near the upper and nasal margins of the optic

disc. The optic discs appeared elevated. The results of a neurologic examination were negative, and pneumoencephalograms were negative for cerebral tumor. The second patient was a man of 35 years who had suffered from frontal headaches for 1½ years and had had attacks of vomiting for two years. The fundi showed bilateral drusen of the nerves with 1.5 D. of papilledema. The visual fields were constricted and the blind spots were enlarged. In this case, also, neurologic and pneumoencephalogram examinations were negative for tumor.

Drusen are probably derived from the neuroglia cells in the optic nerve as a result of some irritative, metabolic, or degenerative process. It is likely that drusen start as minute globules of hyalin, which increase in size by accretion in layers or by fusion. Drusen which are situated anterior to the choroidal ring may present a striking appearance. As the result of their presence the disc may be considerably elevated and may show an increase in diameter. Drusen which are situated deep in the sclerochoroidal canal are probably not visualized with the ophthalmoscope. Such drusen may give abnormal fullness to the disc and enlargement of the blind spot. In many instances, so-called pseudoneuritis may really represent a deeply situated druse. Usually drusen of the optic nerve cause little if any visual impairment, but there are on record cases in which vision was severely affected. In the two cases here described, as well as in other reported cases, the association of severe headaches with drusen has been striking. No adequate explanation for the occurrence of these headaches can be given at present. (4 illustrations, bibliography.)

John C. Long.

13

EYEBALL AND ORBIT

Blaess, M. J. **Cosmetic ocular rehabilitation.** U. S. Naval Med. Bull., 1944, v. 43, July, p. 96.

The author stresses the great importance of the use of an implant in Tenon's capsule, following an enucleation. It is essential that an implant be of proper size, that the capsule and muscles be replaced in anatomic position, and that the maximum amount of conjunctiva be preserved. The writer cites Wiener and Alvis, Allport, Weeks, Clapp, De Schweinitz, and Wheeler in support of the advantages of using an implant. Omission of the implant leads to retraction of the upper lid, tilting of the prothesis, and sagging of the lower lid. Tantalum is suggested as an excellent metal for the implant.

Recent introduction of molded plastic protheses has aided cosmetic rehabilitation. The author also mentions the use of contact lenses over quiet blind eyes. Benjamin Milder.

Walsh, F. B., and Dandy, W. E. **Pathogenesis of intermittent exophthalmos.** Arch. of Ophth., 1944, v. 32, July, pp. 1-10.

Intermittent exophthalmos is a rare but striking and unmistakable syndrome. It is characterized by a pronounced and rapid protrusion of one eye when venous stasis is produced by various positions of the head and neck. The ocular protrusion disappears immediately when the head is erect or when artificially induced venous congestion is relieved. In fact, usually but not invariably, there is enophthalmos when venous congestion does not obtain. Vision may or may not be affected. The condition is progressive. There may be pulsation.

The authors report the case of an 18-year-old girl who had had a typical history for ten years. There was enophthalmos of 3 mm. when the head was erect. The exophthalmos was as follows: 6 mm. when lying recumbent; 11 mm. when the head was tilted forward or the jugular vein was compressed; 18 mm. when expiration was forced and the nostrils were compressed; 5 mm. when the head was turned forcibly to the left, and 3 mm. when it was turned to the right. The eyegrounds were normal.

From study of the case the following conclusions are drawn: (1) The quick protrusion and sinking of the eyeball with the postural changes, and the rapid protrusion induced by coughing, sneezing, and jugular compression could only mean filling of a large venous bed. (2) The pulsation of the eyeball indicates an arterial component. The lesion, therefore, had to be an arteriovenous aneurysm. (3) The absence of a murmur indicates that the communication between arteries and veins was through vascular "coils," and not through a fistula. (4) The enophthalmos (with the patient sitting or standing) is thought to have been due to atrophy of the orbital fat from long-continued pressure.

At operation, a frontotemporal approach to the cranial chamber was made—a typical hypophyseal approach. When the dura was turned back, a large venous mass was found. This was treated by cautery. After surgery the eyeball was free of all the intermittent changes that previously obtained, but the eye was blind, and all the extraocular muscles were paralyzed.

The authors give a complete summary of the condition as reported in the literature. (References, 5 figures, one in color.) R. W. Danielson.

14

EYELIDS AND LACRIMAL APPARATUS

Klunzinger, W. R. **Autoinoculation of the eyelids with vaccinia.** Arch. of Ophth., 1944, v. 31, June, pp. 539-540.

Accidental inoculation of the eyes with vaccinia may occur by transfer of the virus either from the site of vaccination of the patient or from one person to another. The lids, conjunctiva, or cornea may be involved. Corneal lesions have been observed in approximately one third of the reported cases, often with serious visual impairment. In cases resulting from autoinoculation, the sooner the secondary inoculation follows the primary, the more severe and the more prolonged is the secondary reaction.

An eight-year-old boy developed redness and swelling of the lids of both eyes three days after vaccination. The condition of the lids became progressively worse. At the same time a lesion developed on the chin and gradually progressed to a typical vaccinia reaction. The lids of both eyes showed pronounced redness, swelling, and induration, with several marginal ulcerations. There was a moderate mucopurulent discharge. Sulfadiazine, used for a short time, seemed to reduce the fever but had no apparent influence on the lesions, which finally healed without complication. (One photograph, references.) John C. Long.

Mouilie, H., and García Querol, A., **Dacryostomy after resection of the lacrimal sac** (Arruga's operation). Arch. de Oft. de Buenos Aires, 1942, v. 17, Nov., p. 693.

Dacryostomy following Arruga's technique was performed two years after the lacrimal sac had been removed. Drainage of the tears was suc-

cessfully established. The several steps of the operation are described in detail.
Plinio Montalván.

15

TUMORS

González Vanrell, Francisco. **Adenopapilloma of the semilunar fold.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Nov., p. 675.

A case is reported in which a mulberry-like tumor, about 6 mm. in size, was observed in the semilunar fold of the right eye. The tumor was excised and histopathologic examination showed it to be an adenopapilloma. Its rarity is stressed. (References, illustrations, photomicrographs.)
Plinio Montalván.

16

INJURIES

Ayberk, N. F. **Sodoku in the eye.** Göz Klinigi, 1944, v. 1, no. 4, pp. 139-144.

Report of a rare case of sodoku (rat-bite infection) in the eye. A sailor, lying in bed, one night felt a bite in the right upper lid. Ten days later swelling and reddening of the lid started. Then a "phlyctenule" developed, with secondary ulceration. The organs of a mouse infected with the blood of the patient showed typical spirilla. After three injections of neoarsphenamine, the general symptoms (malaise, fever) disappeared and the lid wound healed. (6 illustrations, references.)
Joseph Igersheimer.

Damel, C. S., and Durando, S. A. **Traumatic cysts of the iris, and eyelashes in the anterior chamber.** Arch. de Oft. de Buenos Aires, 1942, v. 17. Oct., p. 614.

The authors report three cases of penetration of lashes into the anterior chamber through perforating wounds of the globe. In the first case the eye was enucleated on account of repeated and violent inflammatory attacks with vision reduced to light perception. In the other two cases implantation cysts of the iris developed and were successfully excised in toto with a small iridectomy at the base of the cyst. The authors believe this procedure to be the treatment of choice. Histopathologic sections were made in the three cases. The subject of cysts of the iris is discussed in detail, with particular emphasis on differential diagnosis. (Illustrations, photomicrographs, references.) Plinio Montalván.

Gallino, J. A. **Obstetrical trauma of the cornea.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Sept., p. 529.

A case is reported in which trauma to the corneas of a new-born was produced by the forceps blades. There was a diffuse opacity the first few days, which cleared up leaving white glassy lines on the cornea as permanent defect. The literature on the subject is reviewed and the pathogenic theories are discussed in detail. (Illustrations, references.) Plinio Montalván.

Meroni, J. C. A. **A case of bilateral cataract following electric shock.** Arch. de Oft. de Buenos Aires, 1942, v. 17, Oct., p. 633.

A 26-year-old man sustained an electric shock produced by a 4400-volt current and two months later presented in the left eye an anterior subcapsular cataract which became mature in a short period of time. One year after the accident similar cataractous changes began to take place in the other eye. The eyes were otherwise essentially normal. Laboratory exam-

inations were negative. Combined extracapsular extraction of the mature cataract was carried out without incident and the final visual result was good. The bibliography on electric cataract is reviewed and the mechanism of production discussed in detail. (References.) Plinio Montalván.

Rosen, Emanuel. **Chalcosis lentis associated with traumatic lenticonus posterior.** Arch. of Ophth., 1944, v. 32, July, pp. 63-65.

Chalcosis lentis has been encountered frequently during the present war because of the employment in armaments of numerous alloys, some containing copper. The toxicity of copper within the globe is well known, and the production of a characteristic "sunflower" just below the anterior capsule of the lens has been regarded as one of the pathognomonic signs of chalcosis lentis. A soldier had received a puncture wound with copper in civilian life, when a 22-mm. rifle backfired and exploded directly into his eye. Several authors are quoted as showing that the copper particles lie in the region of the epithelial cell layer, with deposition in the protoplasm, and with the nucleus of the cell remaining free of the copper. (References.) R. W. Danielson.

Sherman, E. S. **Industrial injuries of the eye.** Arch. of Ophth., 1944, v. 32, July, pp. 33-36.

This paper covers the common-sense rules involved in industrial work. Accurate and complete records and reports are stressed in order to protect both the employee and the employer. (References.) R. W. Danielson.

17

SYSTEMIC DISEASES AND PARASITES

Gordon, B. L. **Ocular tuberculosis, its relation to general tuberculosis.** Arch. of Ophth., 1944, v. 31, June, pp. 541-556.

The first part of this review is devoted to a historical sketch of the development of knowledge of tuberculosis. The observations of Pott, Bichat, Laënnec, Scarpa, Helmholtz, Jaeker, Graefe, Villemin, Pasteur, Koch, and many others are quoted.

The second portion of the review is devoted to the diagnosis of ocular tuberculosis and its relationship to the generalized disease. A number of modern, and often contradictory, views are quoted. The reviewer states that, from the literature cited, the following facts are evident: Tuberculous ocular disease is not nearly so common as generally supposed. Phlyctenular keratoconjunctivitis is not usually of tuberculous origin. The fact that phlyctenular disease has decreased with better living standards, better diet, and the administration of vitamins leads one to think that it is of metabolic origin. Uveal tuberculosis is not necessarily secondary to tuberculosis elsewhere in the body. Bacilli may enter the blood stream and attack the eye directly without pulmonary involvement. The nature of an infection in the anterior segment should be determined in the laboratory. When it is impossible to obtain a specimen of uveal tissue, a presumptive diagnosis may be made by the microscopic appearance of the lesion, by the general status of the patient as to tuberculosis, and by exclusion of other causative factors. Tuberculin tests are of limited value. A negative test may be obtained in the presence of tuberculosis, and a positive test does not determine the location of the tuberculous process. (8 illustrations references.)

John C. Long.

NEWS ITEMS

Edited by DR. DONALD J. LYLE
904 Carew Tower, Cincinnati 2

News items should reach the editor by the twelfth of the month

DEATHS

Dr. John D. Hogue, Altoona, Pennsylvania, died July 19, 1944, aged 62 years.

Dr. Edwin M. Neher, Laguna Beach, California, died July 8, 1944, aged 69 years.

MISCELLANEOUS

The Office of the Surgeon General has designated the following hospitals as special-treatment centers: For plastic surgery and ophthalmologic surgery—Baker General Hospital at Martinsburg, West Virginia; Beaumont at El Paso, Texas; Cushing at Framingham, Massachusetts; Dibble at Menlo Park, California; Northington at Tuscaloosa, Alabama; O'Reilly at Springfield, Missouri; Valley Forge at Phoenixville, Pennsylvania; and Wakeman at Camp Atterbury, Indiana. For ophthalmologic surgery and the blind—Dibble General Hospital at Menlo Park, California, and Valley Forge at Phoenixville, Pennsylvania.

SOCIETIES

At a recent meeting of the Board of Directors of the Academy of Medicine of Cleveland, Dr. A. B. Bruner, Professor of Ophthalmology, Western Reserve University School of Medicine, was elected President of the Academy.

At the ninety-fifth annual meeting of the Indiana State Medical Association held in conjunction with the Army Air Force medical services at the Murat Temple, Indianapolis, Major John G. Bellows (MC) read a paper on "Penicillin in ophthalmology."

Dr. Frederick A. Davis, Madison, Wisconsin, was among the guest speakers at the Inter-State Postgraduate Medical Association of North America assembly at the Palmer House, Chicago, Illinois, October 17th to 20th. The title of his lecture was "What the general practitioner should know about ophthalmoscopic examinations."

The South Carolina Society of Ophthalmology and Otolaryngology held a refresher course from October 31st to November 2d. Among those conducting the course were Dr. Henry M. Goodyear, Cincinnati; Dr. James S. Shipman, Philadelphia; Dr. James W. White, New York; and Dr. Oscar V. Batson, Philadelphia.

At the fifty-fifth annual meeting of the American Pediatrics Society a paper on "Congenital malformations of eyes induced by maternal vitamin-A deficiency" was given by Dr. Josef Warkany and Elizabeth Schraffenberger, Cincinnati.

The Reading Eye, Ear, Nose, and Throat Society of Reading, Pennsylvania, elected the following officers for the fiscal year 1944-45: Dr. Michael J. Penta, president; Dr. R. M. Brickbauer, first vice-president; Dr. Isaac B. High, second vice-president and president-elect; Dr. Harold L. Strause, treasurer; Dr. Paul C. Craig, secretary.

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College of Physicians of Philadelphia, Section on Ophthalmology

CONCERNING THE RELATIONS OF THE DEVELOPING OPTIC NERVE TO THE RECESSUS OPTICUS AND THE HYPOPHYSIS IN YOUNG FOETUSES: A STUDY OF SEVEN HUMAN FOETUSES 4 M.M. TO 40 M.M., INCLUSIVE

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HENRY C. HADEN
Houston

The subject for this study was chosen as it seemed peculiarly appropriate for a lecture in honor of one who had contributed so much to an understanding of the pituitary.

The development of the hypophysis in lower animals has been studied for many years and a very extensive literature upon the subject has been acquired. This had not been so in the case of the human until in comparatively recent years. The description in the books on embryology, either the classics or the most modern, are meager and the illustrations mostly schematic. In these comparatively recent years certain investigators have covered the subject in the human very thoroughly but most of their studies have been made from cross sections and the only representation in their illustrations of the developing optic nerve in relation to the hypophysis is in wax plate reconstructions.[1] A number of years ago, I made a study of the development of the connective tissue frame work of the optic nerve with especial reference to the lamina cribrosa[2] and I have intended supplementing it with a description of the ectodermal frame work. So I had sections made of young foetuses in the horizontal plane of the optic nerve and in addition sagittal and frontal sections of the nerve region of the head. Many of these serial sections included positions in the brain with the developing hypophysis, the optic stalk, nerve, and recessus opticus.

The sections of the head which passed through the horizontal plane of the optic nerve making longitudinal sections of it were kept in the same relation to the optic nerve, whether or not they were above or below the level of it, so they progressed from above downward through the recessus opticus and the third ventricle and then horizontally through the floor of the diencephalon including the eminentia chiasmaticus, tubercinereum, and infundibular recess and then continued down through Rathke's pouch and the primordium of the sella turcica.

In this way they presented the relations of these structures in a somewhat unique fashion. In order to have an intelligent understanding of the parts to be described the antomic details of the region involved should be reviewed. The classical teaching is that the optic vesicle evaginates from the lateral wall of the anterior cerebral vesicle caudally very early and soon afterward the portion of it adjacent to the cerebral vesicle becomes narrow and the hollow stalk develops connecting the optic vesicle with the anterior cerebral vesicle. Shortly afterward the optic vesicle invaginates and forms the optic cup and the invagination extends brainward along the stalk (figs. 4 and 5). The invagination permits the nerve fibers which develop as axons of the neuroblasts in the mantle zone of the innerlayer of the optic cup at about the 14-m.m. age to grow back in the ventral wall of the stalk reaching the brain by the 20-m.m. age. At the sites of the optic evaginations from the brain there is a recess or furrow in the floor of the anterior cerebral vesicle passing from one optic vesicle to the oth-

* Delivered before the College of Physicians, Section on Ophthalmology, Philadelphia, December 16, 1943.

er, called the recessus opticus (fig. 1). The teaching of certain embryologysts is that the fibers of the optic nerve grow into the marginal zone of the optic recess and fill it, obliterating its lumen and thus form the optic chiasm. It is part of this thesis to show that although the fibers grow into the marginal zone of the recessus opticus they do not obliterate its lumen and when they reach the region of the diencephalic floor they turn slightly caudally and enter the eminentia chiasmaticus into which they pass forming the primordium of the optic chiasm.[3,4,5] The lumen of the recessus opticus stays open after the chiasm has begun to have its classical appearance,[6] and remains as the most anterior recess in the floor of the third ventricle in post natal life.[7] To be properly oriented as to the location of the parts under discussion a study should be made of a sagittal section through the middle of the forebrain of a young embryo before the epithelial walls have begun to differentiate. The brain at the 4 m.m. age, the age at which this study begins, is divided into the three primary cerebral vesicles, prosencephalon, mesencephalon, and rhombencephalon. The section illustrated, a median sagittal one (fig. 1), leaves the middle of the neural tube just caudad to the anterior cerebral vesicle, the prosencephalon, but the prosencephalon is well seen. The ectodermal cells of its walls show no differentiation but contain many mitotic figures. The forebrain is flexed so as to be nearly parallel with the hindbrain and the body of the embryo. The anterior neuropore has not completely closed (fig. 1), and caudad to it is a stretch of wall, the lamina terminalis, which ends at a recess, the recessus opticus (fig. 1).[8] A line drawn almost vertically from behind the optic recess across the prosencephalon to the site of the future paraphysis will divide the prosencephalon into the telencephalon, the most anterior part of the brain, and the diencephalon or interbrain, caudad to the line.[3] This places the recessus opticus in the telencephalon. The recessus opticus remains in the telencephalic portion of the third ventricle post natally. The lamina terminalis lying between the anterior margin of the optic recess and the anterior neuropore forms the lower part of the anterior wall of the third ventricle (fig. 1). The other special divisions of the floor of the third ventricle have not appeared at this age but their positions can be indicated which facilitates an understanding of its later development. All the structures in the floor of the third ventricle caudad to the optic recess are in the diencephalon. They are the eminentia chiasmaticus, tubercinereum, infundibular recess, and mamillary recess (fig. 1). The diencephalic floor is one of the most vital portions of the brain and I believe that obscure eye symptoms have their origin here besides those produced by neoplasm. The diencephalic floor differs very much in these young foetuses from the developed human and also from the older foetuses. This thesis concerns only those through the 40 m.m. age. When the infundibulum evaginates from the diencephalic floor (the floor of the third ventricle) it is not immediately caudad to the optic chiasm, as is often described,[9] for the reason that the optic chiasm does not exist at that time. The eminentia chiasmaticus which lies immediately posterior to the optic recess indicates the position of the future chiasm and the evaginating infundibulum is quite some distance caudad to it and continues so until late in foetal life. The infundibulum evaginates about the 7 m.m. age and at the 8 m.m. age it is a short protrusion with a round knoblike extremity (fig. 3). As it grows and the stalk (stem) and infundibular process develop they extend backward horizontally and remain nearly parallel to the floor of the

third ventricle (diencephalic floor) until near the 40 m.m. age when they begin to show some downward tendency. The angle between the diencephalic floor and the infundibular stem (stalk) increases with the age of the foetus and the distance between the infundibulum and the location of the optic chiasm decreases as the angle increases.[1] At birth the infundibular stem is in close relation with the optic chiasm and it is practically perpendicular to the diencephalic floor. Below the floor of the diencephalon will lie the epithelial (Buccal) portion of the hypophysis. At this age it is only indicated by an evagination from the superior posterior wall of the stomodeum (fig. 1).[10] Systematic writers have divided the hypophysis into an anterior epithelial portion derived from Rathke's pouch, a diverticulum from the epithelial wall of the stomodeum, and a nervous posterior portion derived from the floor of the diencephalon. The epithelial portion is subdivided into three parts, pars anterior (pars distalis), pars intermedia (pars infundibularis), and pars tuberalis. The neural portion, pars nervosa, is usually divided into the infundibular stalk and infundibular process. Tilney is more detailed and divides it into the median eminence of the tuber cinereum, the infundibular bulb, infundibular stem, and infundibular process.[1]

It has been taught that quite early in foetal life a diverticulum appears in the posterior superior wall of the stomodeum, Rathke's pouch (figs. 1 and 2), and an evagination, from the diencephalic floor, the infundibulum, grows down to meet it (fig. 3). When it does the pouchlike diverticulum becomes concave dorsally and two hornlike processes, the tuberal processes, grow up to either side of the infundibulum (figs. 6 and 7). This theory has been denied by certain investigators. It is not in the province of this paper to discuss this. However, I may state that there are numerous mitotic figures in the cells of the wall of the anterior cerebral vesicle of the 4 m.m. embryo in the region of the future diencephalic floor and that the epithelial wall of the stomodeum seen in median sagittal sections is not in contact with the vesicle wall. In para transverse sections of an 8 m.m. embryo Rathke's pouch is seen as a flat oval (fig. 2) lying below the forebrain. Some sections caudad, the primordium of the infundibulum, which is short and ends in a knoblike process, is seen approaching (fig. 3) Rathke's pouch, which is not changed from the oval shape and is not in contact with the floor of the brain.

In embryos of the 10 m.m. age Rathke's pouch has become concave dorsally under the approaching infundibulum and presents the tuberal processes (figs. 6 and 7). The infundibulum is some distance caudad to the optic stalks.

In foetuses of the 20 m.m. age much more detailed development has occurred. The head of the 20 m.m. foetus illustrated was bisected in the middle in order to show horizontal sections of one optic nerve and cross sections of its fellow (fig. 8). Fortunately for this study the bisection passed well to one side of the middle and thus the structures in the center of the brain were included in the transverse sections (fig. 8, those cutting the optic nerve in a longitudinal direction). In them some fibers from the ganglion cells of the retina are seen to have grown back as far as the brain, and have passed through the inferior posterior wall of the optic recess into the eminentia chiasmaticus. When seen superior to the level of the nerve the optic recess is a wide open space communicating with the third ventricle (fig. 9). At a little lower level the central portion of the lumen of the optic recess disappears because the sections have passed below it (fig. 10). At this level either end of the optic recess

tapers (fig. 10) to join the lumen of the optic stalk which is still patent in the posterior third of the optic nerve (fig. 11 higher magnification; fig. 12 higher magnification, cross section of the fellow nerve in its posterior portion). Below the level of the patent lumen the glial nuclei in the nerve are arranged irregularly, not in definite rows (fig. 13), and among them pass the nerve fibers which enter the floor of the brain, below and caudad to the lumen of the optic recess, and pass into the eminentia chiasmaticus in the floor of the diencephalon (figs. 13 and 14). Posterior to the eminentia is the tuber cinereum with which it is continuous. In the caudal portion of the tuber cinereum is a lozenge shaped opening (fig. 14 higher magnification of fig. 13) leading into the infundibulum, flanked on either side by the tips of the tuberal processes of Rathke's pouch (fig. 14 higher magnification). At a lower level the tuberal processes, into which the lumen of Rathke's pouch extends, lie under and in contact with the floor of the diencephalon and between them is the infundibular process (figs. 15, 16 higher magnification). The infundibular process is located some distance caudad to the primordium of the optic chiasm. Below this level lies the typically shaped Rathke's pouch (fig. 17) with large lumen and tuberal processes and the primordium of pars intermedia (pars infundibularis) and infundibular process. Caudad to this is a condensed area of mesenchyme, the primordium of the dorsum sella (fig. 17). As the sections pass lower the lumen of the pouch disappears and its ventral wall is seen (fig. 18).

Twenty-four m.m. age. The optic recess communicates with the third ventricle through a wide opening (fig. 20). Below the level of its middle portion the sections pass into its floor (fig. 21) but laterally the lumen is open and the recess tapers to the optic nerves as in the 20 m.m. age. Below the level of the third ventricle lie the chiasma primordium (into which the optic nerve fibers have begun to grow) and the tuber cinereum in the diencephalic floor (fig. 22). In the caudal portion of the tuber cinereum there is an opening similar to the one in the 20 m.m. foetus which leads into the infundibulum. The infundibular opening is quite some distance caudad to the primordium of the optic chiasm (fig. 22). At a lower level the optic nerve fibers have entered the chiasma primordium which is continuous with the tuber cinereum (fig. 23). Under the caudal portion of the tuber cinereum lie the infundibular stem and process (figs. 23 and 24) flanked by the tuberal processes of Rathke's pouch into which the lumen of the pouch extends (fig. 24). Below the level of the diencephalic floor lies the typically shaped Rathke's pouch together with the infundibular process and pars intermedia (fig. 25). Some sections lower than this the tips of the tuberal processes are curved more toward forming the anterior portion of the pars anterior (distalis; fig. 26). The remains of the stalk is patulous at this level. At a still lower level the tips of the processes are connected by a series of small epithelial rosettes or acini (fig. 27), and caudad to the infundibular process is a condensed area of mesenchyme, the primordium of the dorsum sella.

In the 29 m.m. age the optic nerve showed much more development as regards the nerve fibers but the glial cells were not lined up in regular columns dividing the funiculi (fig. 28; the illustrations of the 29 m.m. age foetus are of half of a head which had been bisected in the middle). The fibers had grown into the chiasma eminence, posterior to which and continuous with it was the median eminence of the tuber cinereum with its

horizontally extended cavity (fig. 28). Above the level of the nerve fibers (fig. 29) the lumen of the optic recess was open and communicated with the third ventricle which was continuous with the horizontal cavity[1] of the median eminence of the tuber cinereum. When seen in cross section the optic nerve of the fellow eye, after it had entered the cranium, presented a lumen in its upper portion with a zone of nuclei above and a broader zone of fibers below (fig. 30 higher magnification). Farther inward the lumen of the nerve passed into the lumen of the recessus opticus (fig. 31 higher magnification), below the lumen of which the fiber zone was much reduced in size (fig. 31 higher magnification). Deeper in under the tuber cinereum the epithelial hypophysis was seen in half section, its tuberal process reaching nearly to the median eminence (fig. 32 higher magnification). The remains of the stalk of Rathke's pouch with a small lumen was seen. Sections farther down showed no lumen in the stalk which was reduced to a solid cord of epithelial cells.

In a foetus of the 40 m.m. age, sectioned at a level above the optic nerve and chiasm, the optic recess was seen to be widely open and communicating with the third ventricle (fig. 33). The third ventricle at this age is a narrow slitlike cavity whose walls are in contact in the central portion (fig. 34 higher magnification). At a lower level the optic nerve fibers were seen to enter the chiasm caudad to the optic recess (fig. 35 higher magnification) and when the sections reached a deeper level the lumen of the optic recess disappeared from view (fig. 36 higher magnification) not because it had been filled with nerve fibers but because the sections had passed below it and lay in the floor of the diencephalon. Caudad to the chiasm lay the tuber cinereum with which it was continuous (fig. 36 higher magnification). At a still lower level and some distance caudad to the optic chiasm there was a lozenge-shaped opening in the tuber cinereum (fig. 37 higher magnification) similar to the one in the younger foetuses, the infundibulum (infundibular bulb). It was partially flanked by the tuberal processes as it was in the younger foetuses. At a lower plane below the floor of the tuber cinereum the infundibular stalk passes into the infundibular process (fig. 38 higher magnification). The latter is partially surrounded by the pars intermedia which shows glandular arrangement. Below this level the lateral portions of the tuberal processes which had had large cavities continuous with the lumen of Rathke's pouch in the younger foetuses, were filled with epithelial cells, acini, and mesenchyme to form the pars distalis (fig. 39 higher magnification) while their medial parts remain in contact with the tuber cinereum and form the pars tuberalis (figs. 39 and 40 higher magnification). At a lower level (fig. 41) the sections passed below the floor of the brain and through the primordium of the sella turcica (fig. 41) in which lay the pars distalis separated from the pars intermedia by the residual lumen of Rathke's pouch and caudad to and in contact with the pars intermedia was the infundibular process. These will be the contents of the sella turcica, covered by the diaphragma sella postnatally.

The study of these foetuses has revealed a wealth of interest in the developing mesenchyme and details of the histology of the hypophysis itself but this thesis pertains to morphology and topography alone. It is an endeavor to present the recessus opticus in a more rational light and to show the relation of the developing optic nerve, chiasm and hypophysis in early foetal life.

References

[1] Tilney, Frederick. The human hypophysis. Bul. Neurological Inst. of New York, 1936, v. 5.

[2] Haden, Henry C. The development of the connective tissue framework of the human optic nerve, with especial reference to the lamina cribrosa. Amer. Jour. Ophth., 1925, v. 8.

[3] Bailey and Miller. Text-book of embryology. Ed. 4, (von Kupffer) Scheme, fig. 364, pp. 425, 501.

[4] Cunningham's Text-book of anatomy, p. 553.

Salzmann, Maximilian. Anatomie und Histologie des menschlichen Augapfels.

Bach, Ludwig, und Seefelder, R. Atlas zur Entwicklungsgeschichte des menschlichen Auges.

Wright, Sampson. Applied physiology. Ed. 4.

Keibel and Mall. Manual of human embryology.

[5] Tilney, Frederick. The morphology of the diencephalic floor. Jour. Comp. Neurol., 1915, v. 25.

Gilbert, Margaret Shea. The development of the hypophysis. Amer. Jour. Anat., 1934, v. 54.

Kingsbury, B. F., and Roemer, F. J. The development of the hypophysis of the dog. Amer. Jour. Anat., 1940, v. 66.

[6] Frazer, J. Ernest. A manual of embryology. Ed. 2.

Minot, C. S. Human embryology.

Engelbach, William. Endocrine medicine, v. 1.

Duke-Elder, Sir W. Stewart. Text-book of ophthalmology, v. 1.

[7] Sobotta-McMurrich. Atlas and text-book of human anatomy. v. 3, fig. 648, pp. 133, 155, 156.

[8] His, Wilhelm. Ueber das frontale Ende des Gehirnrohres. Separat-Abzug aus Archiv für Anatomie und Physiologie. Anatomische Abtheilung, 1893.

[9] Jordan and Kindred. Text-book on embryology. Ed. 4, p. 350.

Da Costa. Gray's anatomy. 1905, pp. 882, 902.

Atwell, Wayne J. Development of hypophysis cerebri in man, with special reference to the pars tuberalis. Amer. Jour. Anat., 1926, v. 37.

Wislochki, George B. The meningeal relations of the hypophysis cerebri. Amer. Jour. Anat., 1937, v. 61.

Mann, Ida C. The development of the human eye.

[10] His, Wilhelm. Anatomie menschlicher Embryonen, v. 3, figs. 2 and 3, p. 16.

His, Wilhelm. Vorschläge zur Eintheilung des Gehirns. Separat-Abzug aus Archiv für Anatomie und Physiologie. Anatomische Abtheilung, 1893.

These illustrations were made from unretouched photographs of sections of human embryos and foetuses in my private collection.

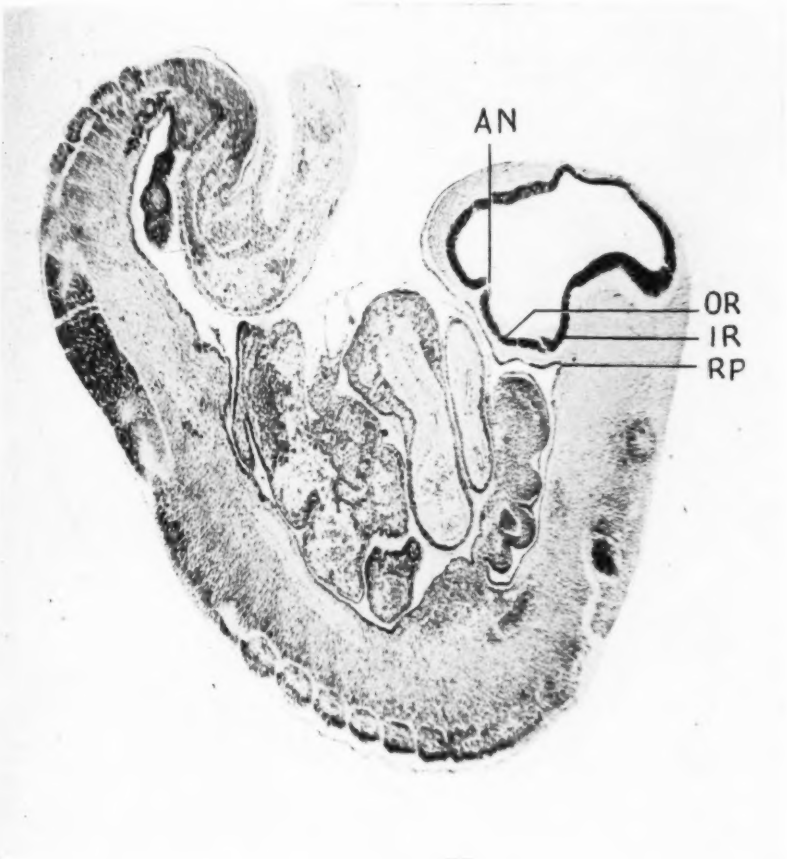

Fig. 1 (Haden). Median sagittal section of 4 m.m. embryo passing through anterior cerebral vesicle (prosencephalon). AN, anterior neuropore; OR, recessus opticus; IR, position of infundibular recess; section of floor of vesicle from (AN to OR) is lamina terminalis. Caudad to OR is diencephalic floor. Immediately caudad to OR is chiasma eminence. RP, evagination from stomodeum, Rathke's pouch. There is no mesoderm between the floor of the vesicle and the surface ectoderm between the anterior neuropore and Rathke's pouch.

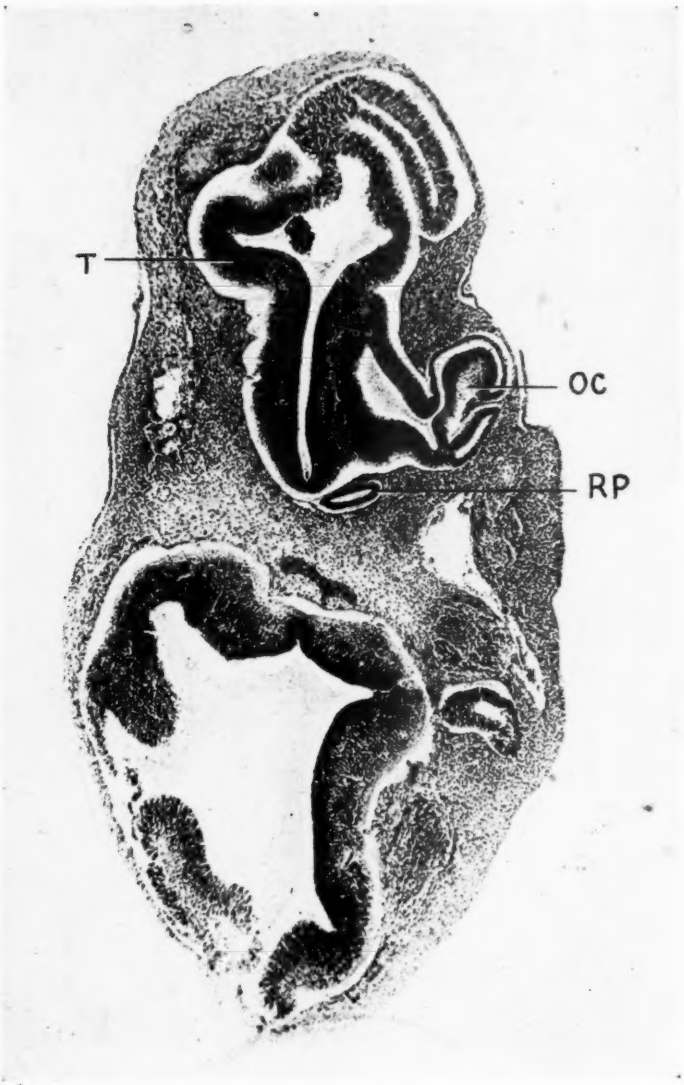

Fig. 2 (Haden). Paratransverse section of head of 8 m.m. embryo. T, telencephalon; OC, optic cup; RP, Rathke's pouch below brain.

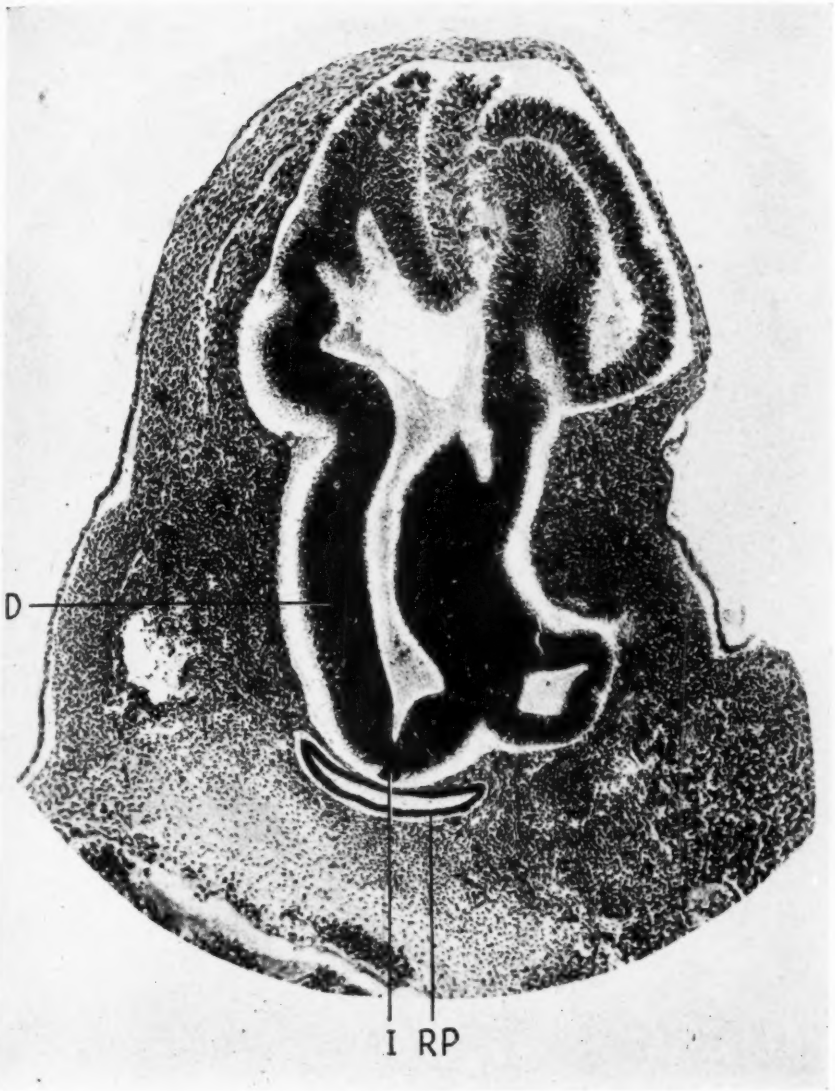

Fig. 3 (Haden). Paratransverse section of head of 8 m.m. embryo. Caudad to figure 2, higher magnification. D, diencephalon; I, infundibulum with knoblike extremity below which is RP, Rathke's pouch.

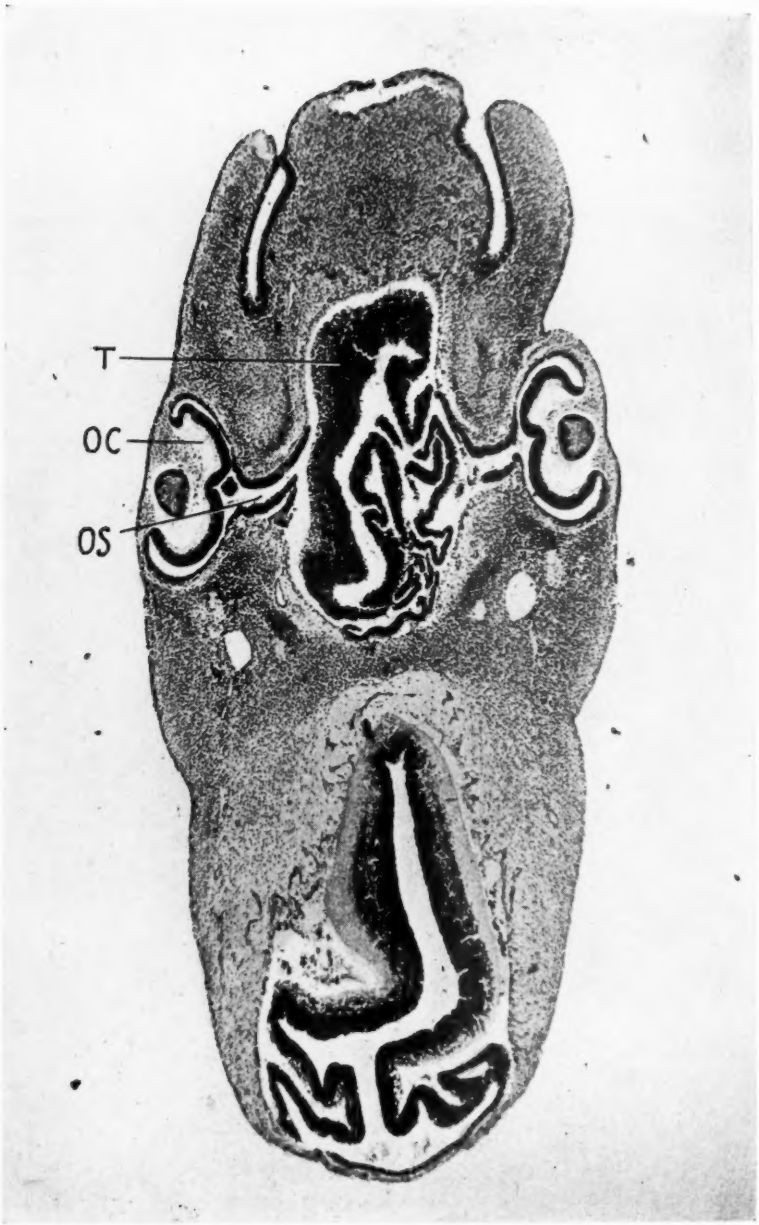

Fig. 4 (Haden). Transverse section of head of 10 m.m. embryo. T, telencephalon; OC, optic cup; OS, optic stalk.

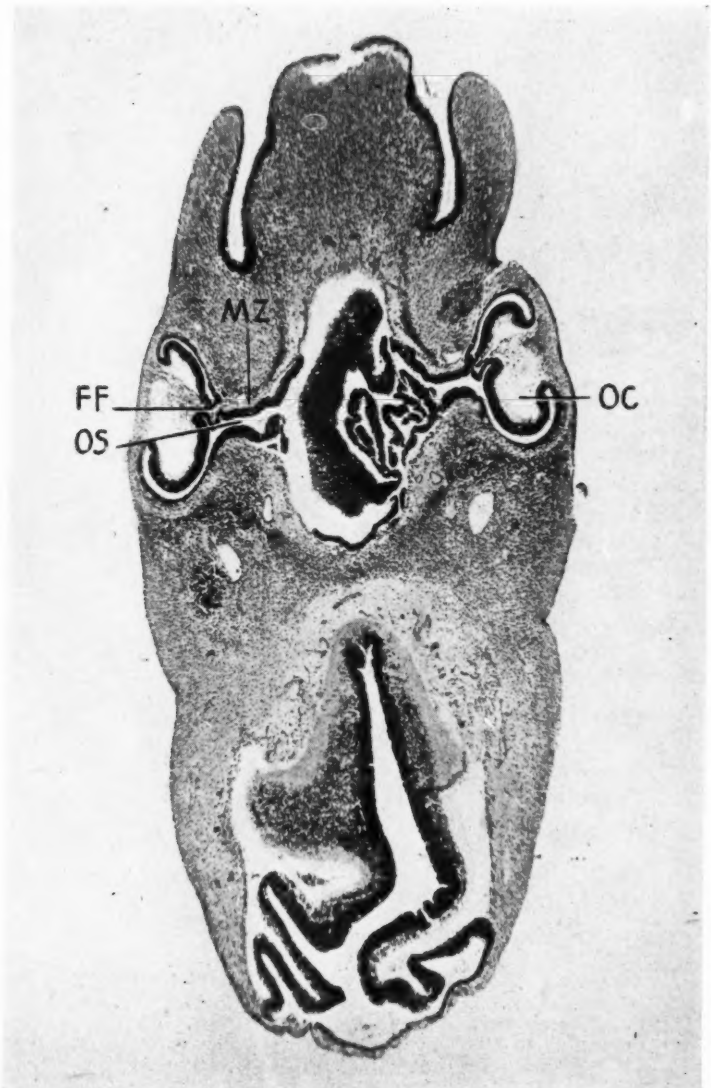

Fig. 5 (Haden). Transverse section of head of 10 m.m. embryo. Lower level than figure 4. OC, optic cup; OS, optic stalk; FF, foetal fissure; MZ, marginal zone of stalk wall.

Fig. 6 (Haden). Transverse section of 10 m.m. embryo lower level than figure 5. TV, diencephalic portion of third ventricle which extends into, I, infundibulum, primordium of stem and process; TP, Rathke's pouch with tuberal processes; D, diencephalon.

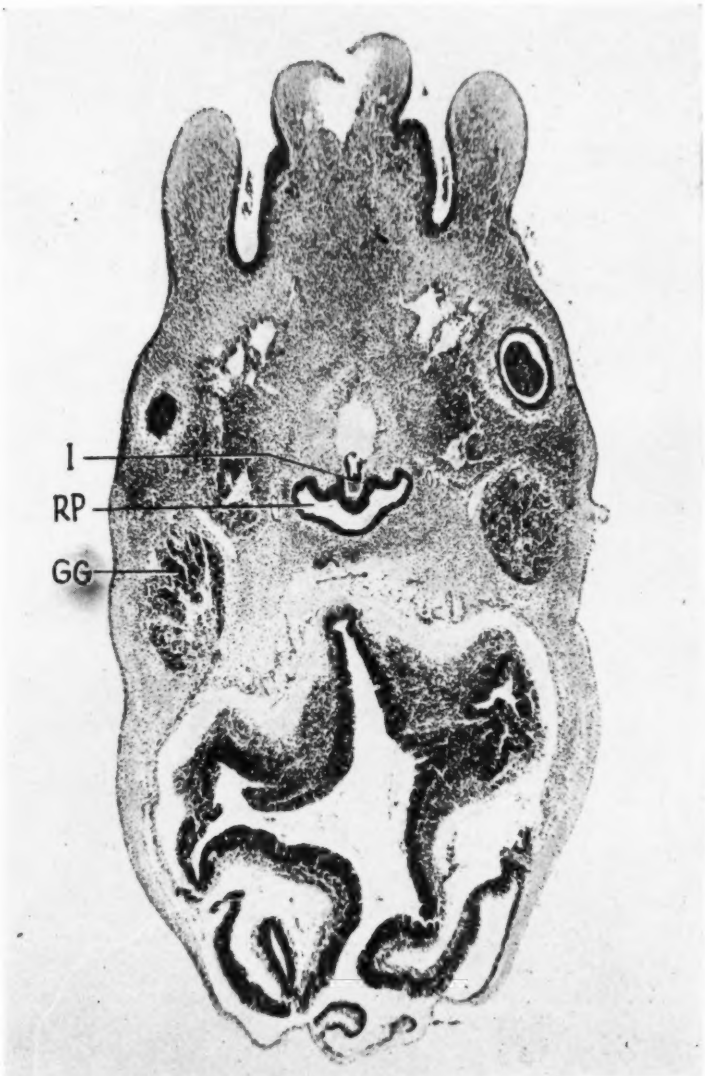

Fig. 7 (Haden). Transverse section of 10 m.m. embryo lower level than figure 6 below diencephalic floor. I, tip of infundibulum and RP, Rathke's pouch; GG, Gasserian ganglion.

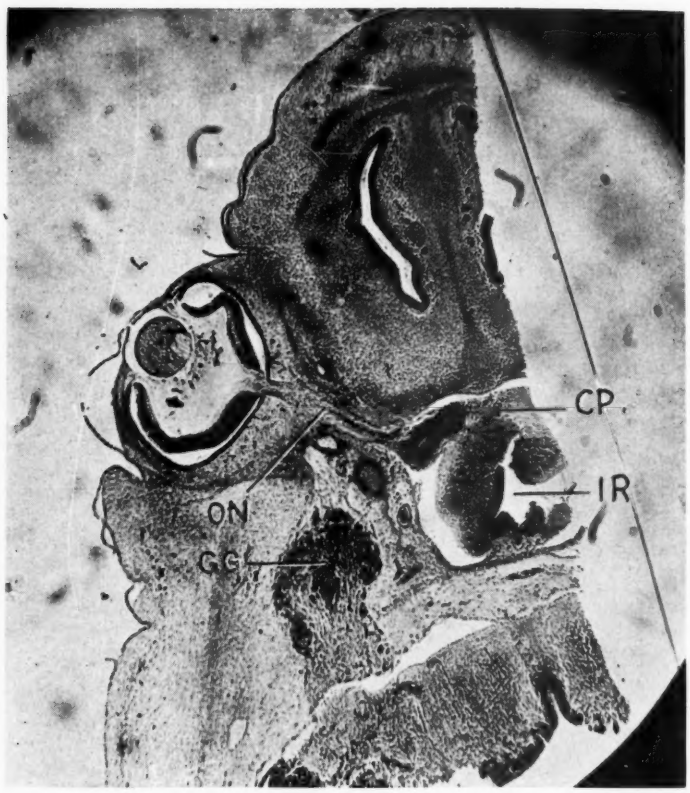

Fig. 8 (Haden). Transverse section of 20 m.m. foetus passing horizontally through optic nerve. The head was bisected but bisection passed well to side of middle. ON, optic nerve; CP, chiasma primordium; IR, infundibular recess of third ventricle; GG, Gasserian ganglion.

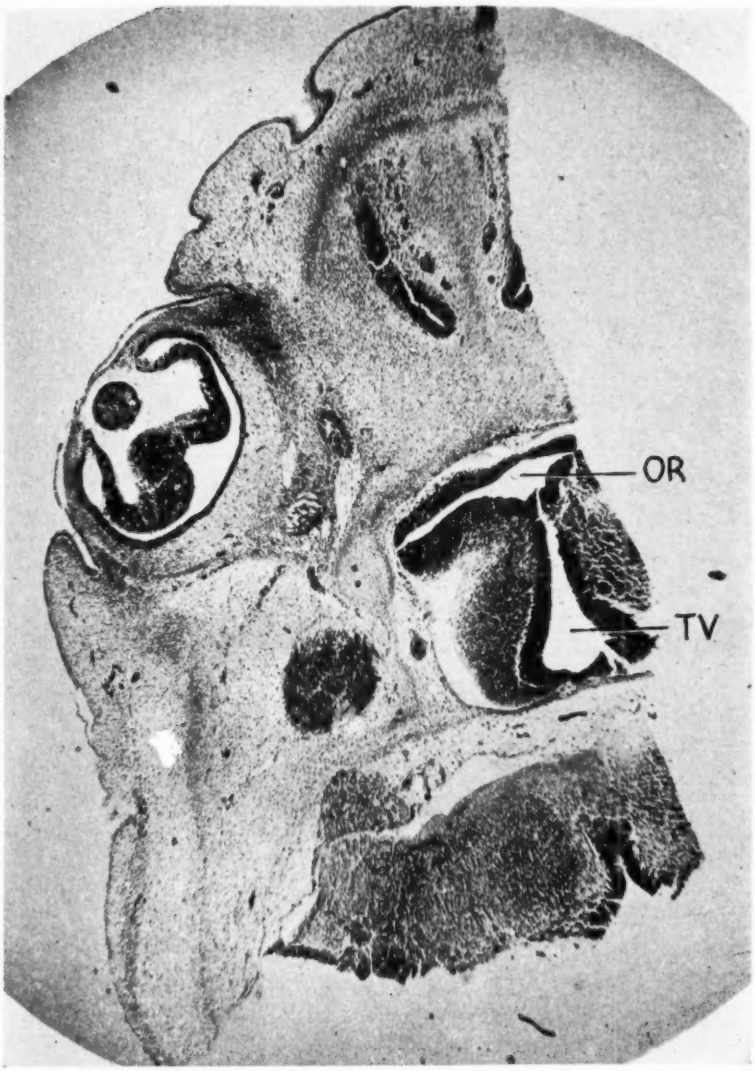

Fig. 9 (Haden). Transverse section of 20 m.m. foetus at higher level than figure 8 but in the same plane. OR, optic recess communicating with the TV, third ventricle.

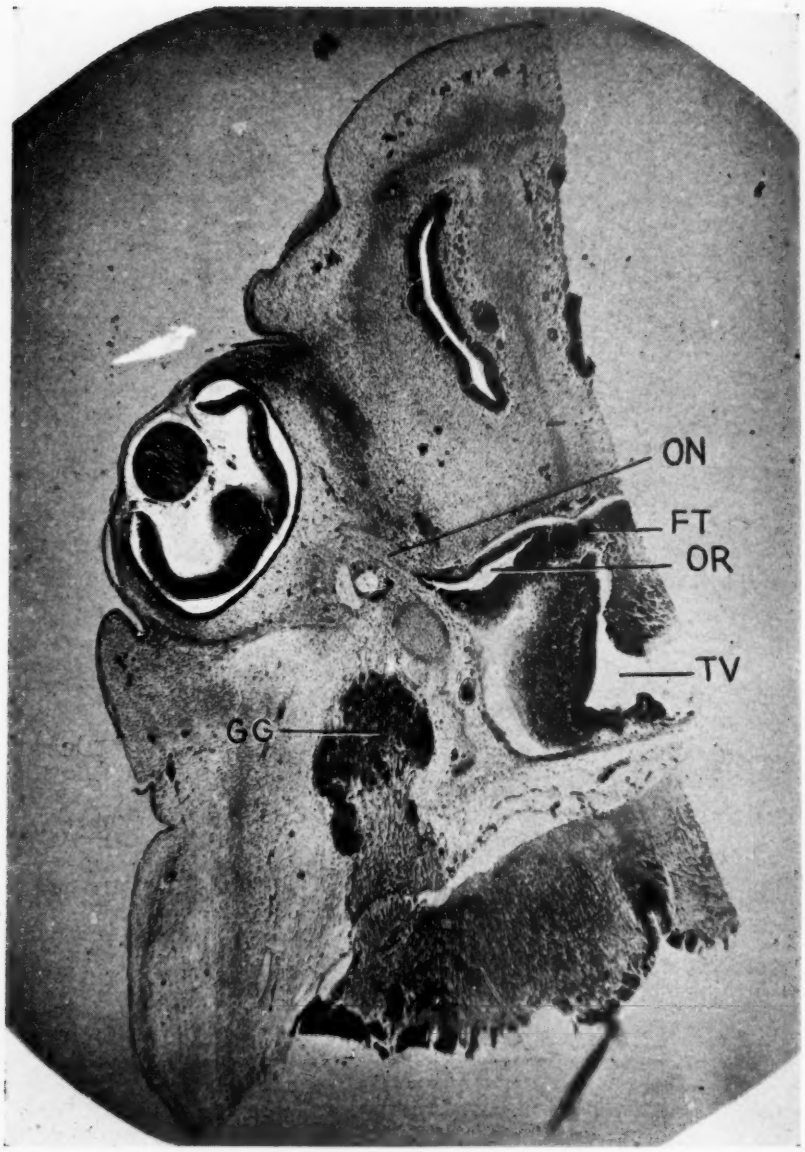

Fig. 10 (Haden). Transverse section of 20 m.m. foetus at slightly lower level than figure 9 but in the same plane. OR, optic recess either end of which tapers to ON, optic nerve, and is continuous with its residual lumen; FT, section passes below lumen of central portion of optic recess and lies in telencephalic floor. TV, third ventricle; GG, Gasserian ganglion.

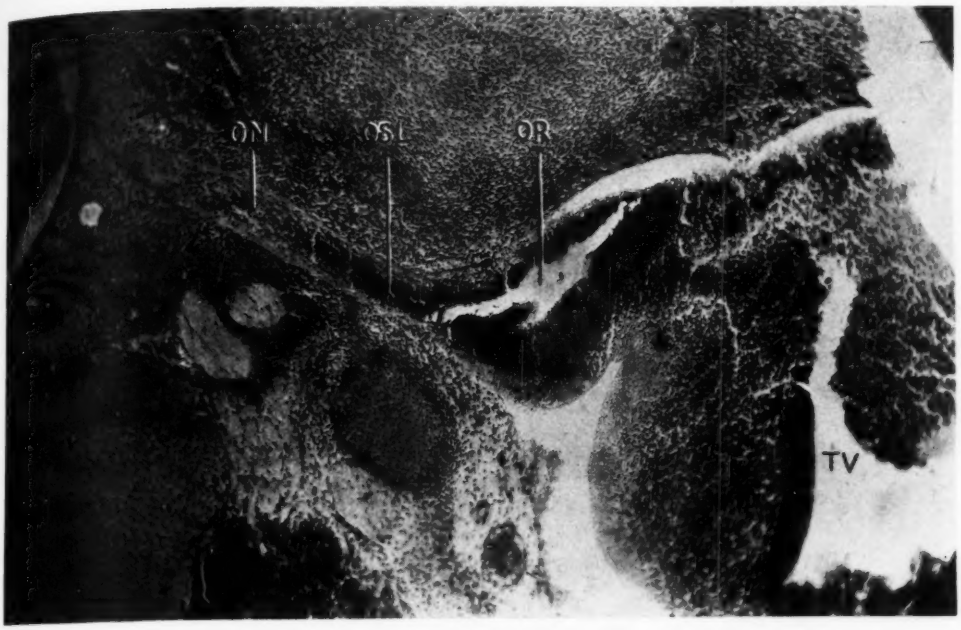

Fig. 11 (Haden). Transverse section of 20 m.m. foetus at a lower level than figure 10 but in the same plane. Higher magnification. OSL, the residual optic stalk lumen is open in the posterior third of ON, optic nerve, and communicates with OR, optic recess; TV, third ventricle.

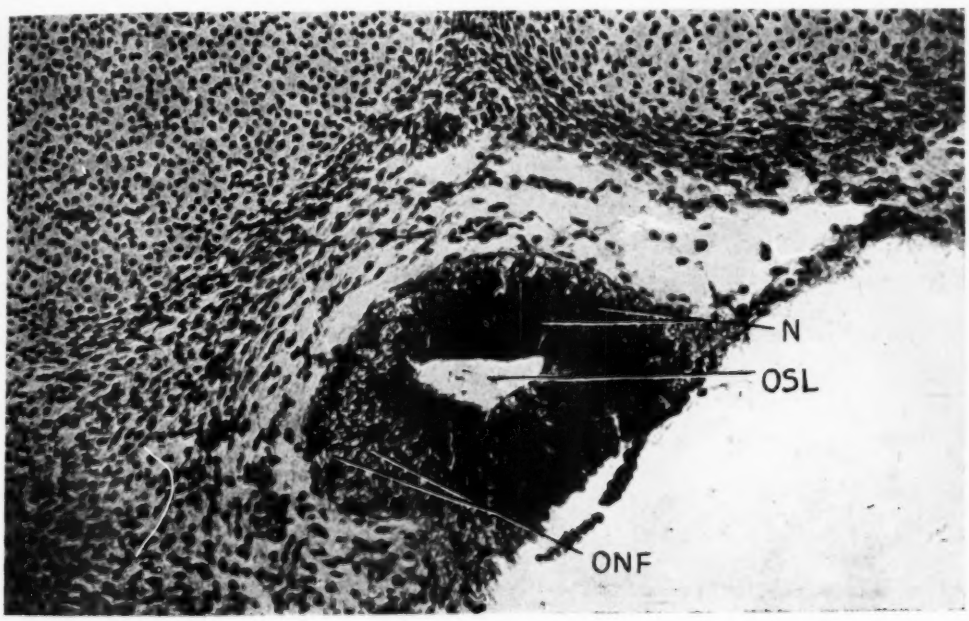

Fig. 12 (Haden). Cross section of the fellow nerve of the 20 m.m. foetus in its posterior third. Higher magnification. N, nuclei; OSL, optic stalk lumen; ONF, optic nerve fibers.

Fig. 13 (Haden). Transverse section of 20 m.m. foetus same plane as figure 11 but below level of lumen of nerve. ON, optic nerve, fibers of which enter floor of OR, optic recess, and turn caudally into CE, chiasma eminence. TC, tuber cinereum with lozenge-shaped opening in its posterior portion, the I, infundibular recess.

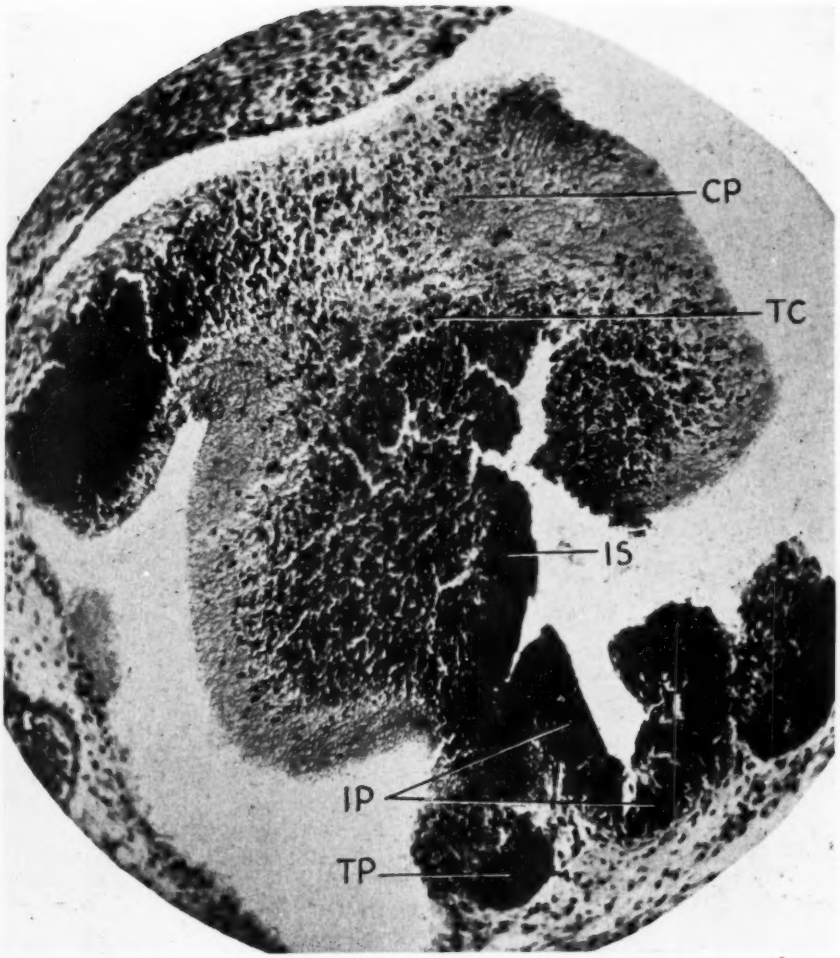

Fig. 14 (Haden). Higher magnification of posterior portion of figure 13. CP, chiasma primordium (chiasma eminence); optic nerve fibers above on right turn caudally to enter chiasma eminence; TC, tuber cinereum; section passes obliquely through IS, infundibular stalk (stem) and IP, infundibular process. TP, tip of tuberal process of Rathke's pouch.

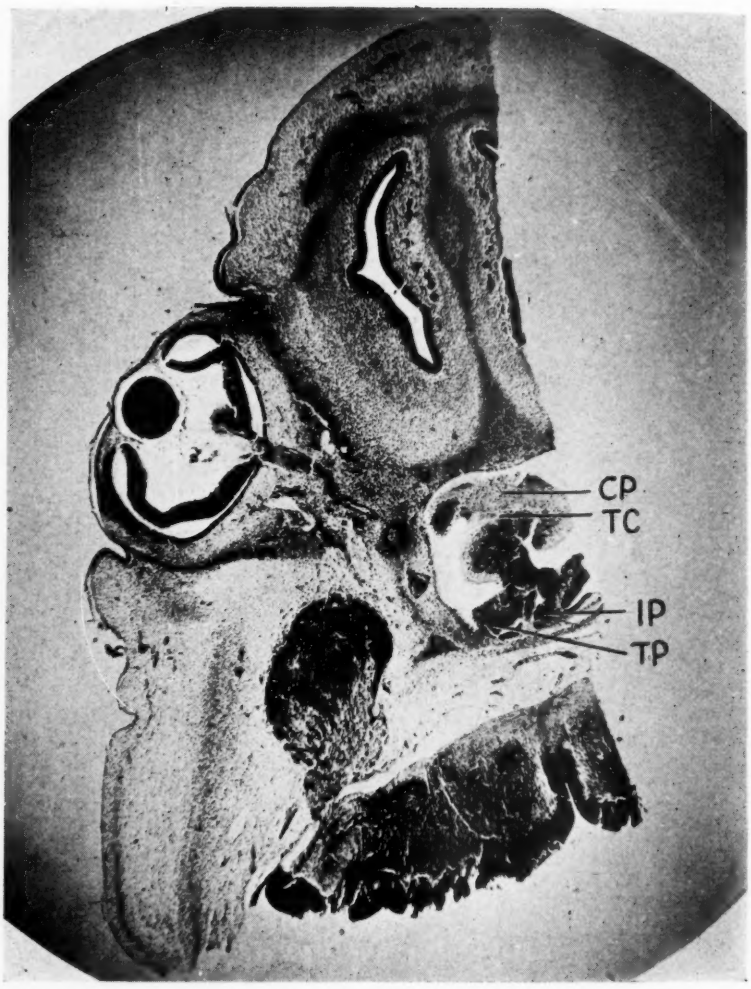

Fig. 15 (Haden). Transverse section of 20 m.m. foetus at lower level than figure 13 but in the same plane. CP, chiasm primordium; TC, tuber cinereum; IP, infundibular process flanked by TP, tuberal processes of Rathke's pouch with lumen.

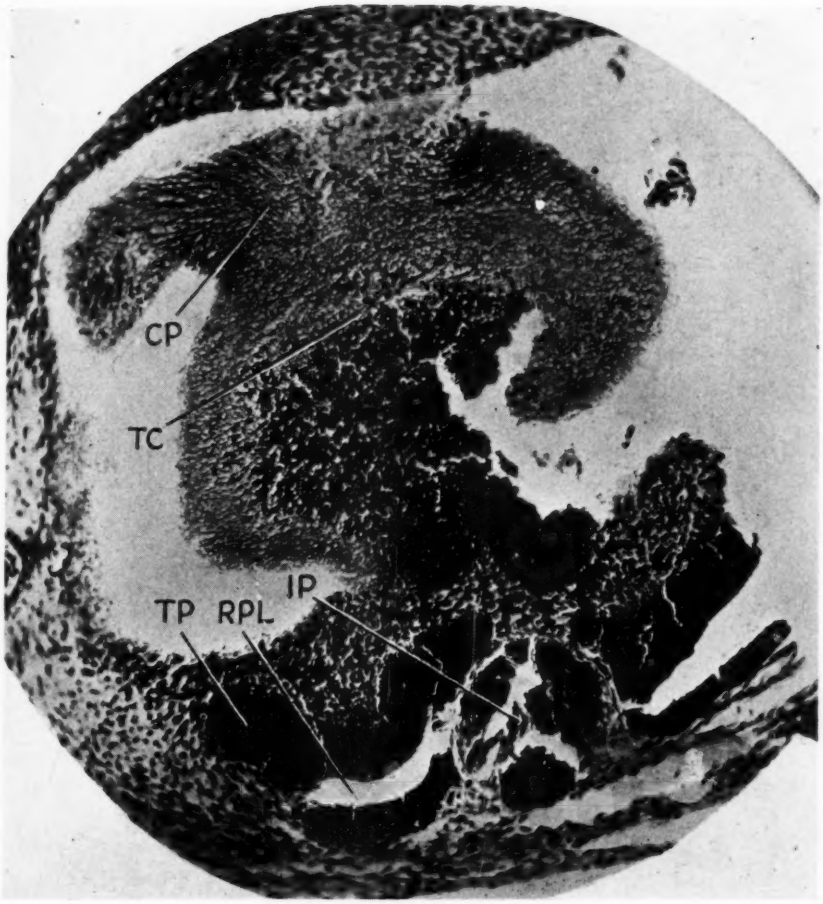

Fig. 16 (Haden). Transverse section of 20 m.m. foetus; higher magnification at slightly lower level than figure 15. CP, optic nerve fibers in chiasm primordium; TC, tuber cinereum; IP, infundibular process; TP, tuberal processes; RPL, Rathke's pouch with lumen.

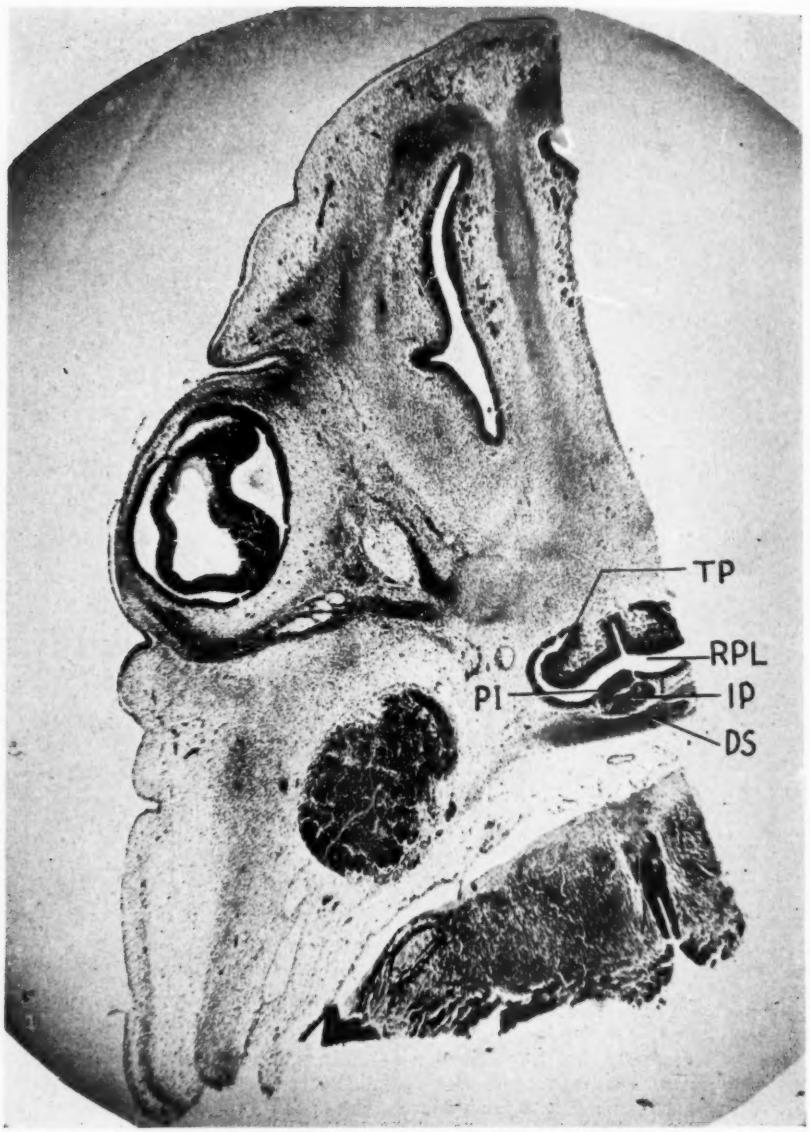

Fig. 17 (Haden). Transverse section of 20 m.m. foetus at lower level than figure 16 but in the same plane. RPL, lumen of typically shaped Rathke's pouch; TP, tuberal process; PI, pars intermedia (pars infundibularis); IP, infundibular process; DS, dorsum sella primordium.

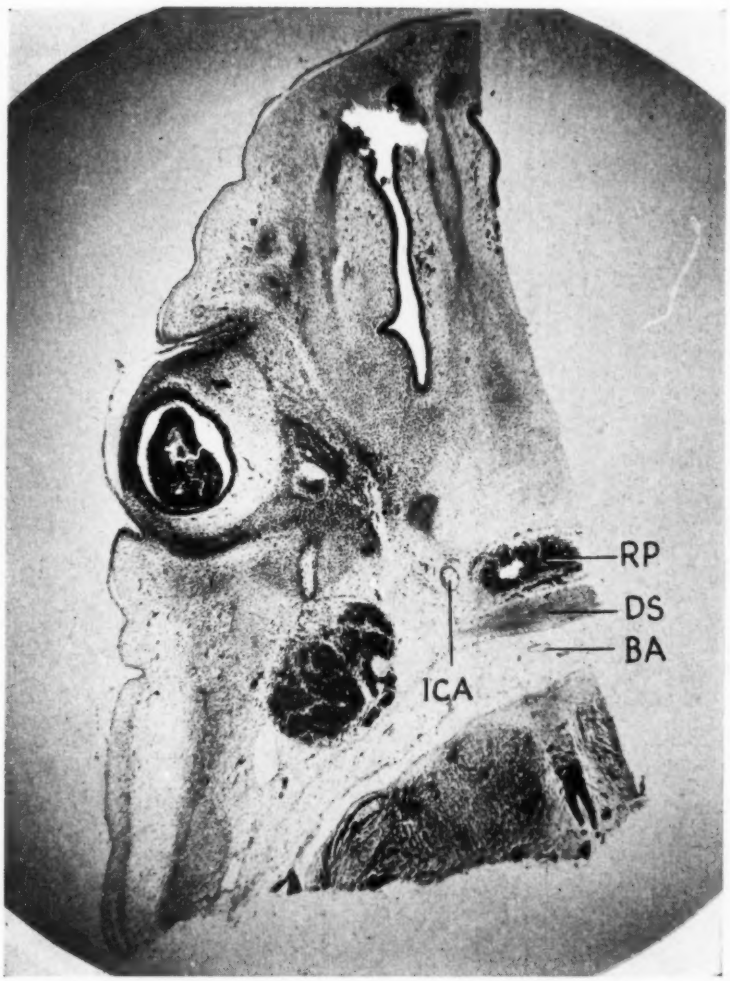

Fig. 18 (Haden). Transverse section of 20 m.m. foetus at slightly lower level than figure 17 but in the same plane. Inner surface ventral wall of RP, Rathke's pouch; DS, dorsum sella primordium; BA, basilar artery; ICA, internal carotid artery.

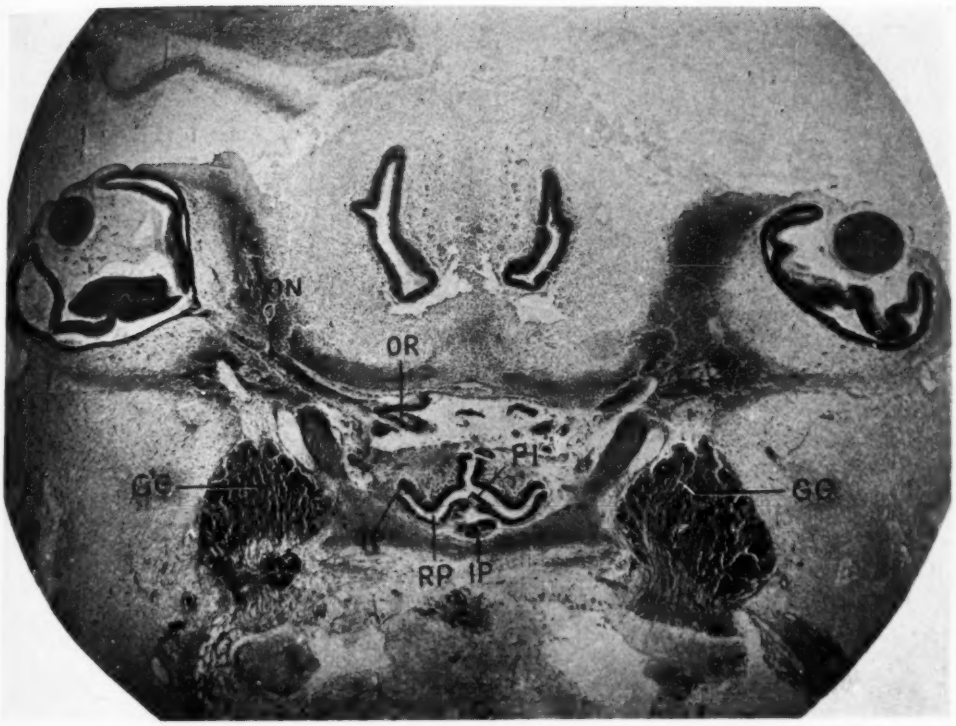

Fig. 19 (Haden). Transverse section of head of 18 m.m. foetus. ON, optic nerve; OR, optic recess; RP, Rathke's pouch; IP, infundibular process; PI, pars intermedia; TP, tuberal process; GG, Gasserian ganglion.

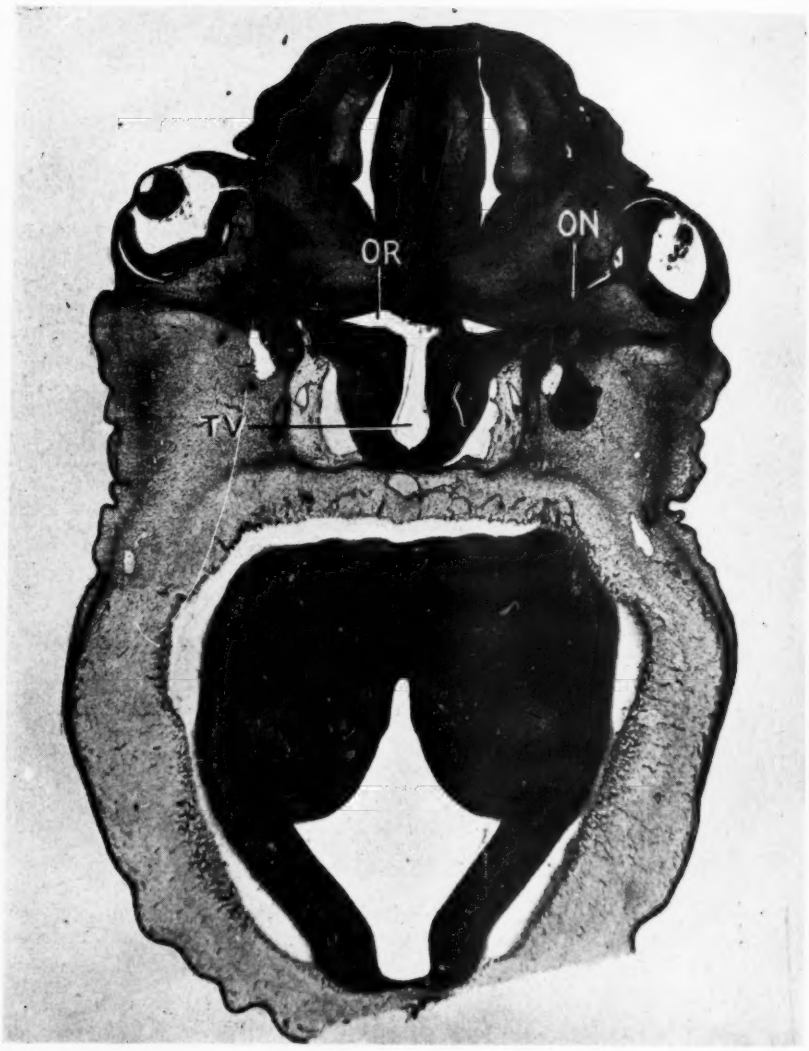

Fig. 20 (Haden). Transverse section of 24 m.m. foetus. In the plane of the ON, optic nerves; OR, optic recess communicating with the TV, third ventricle.

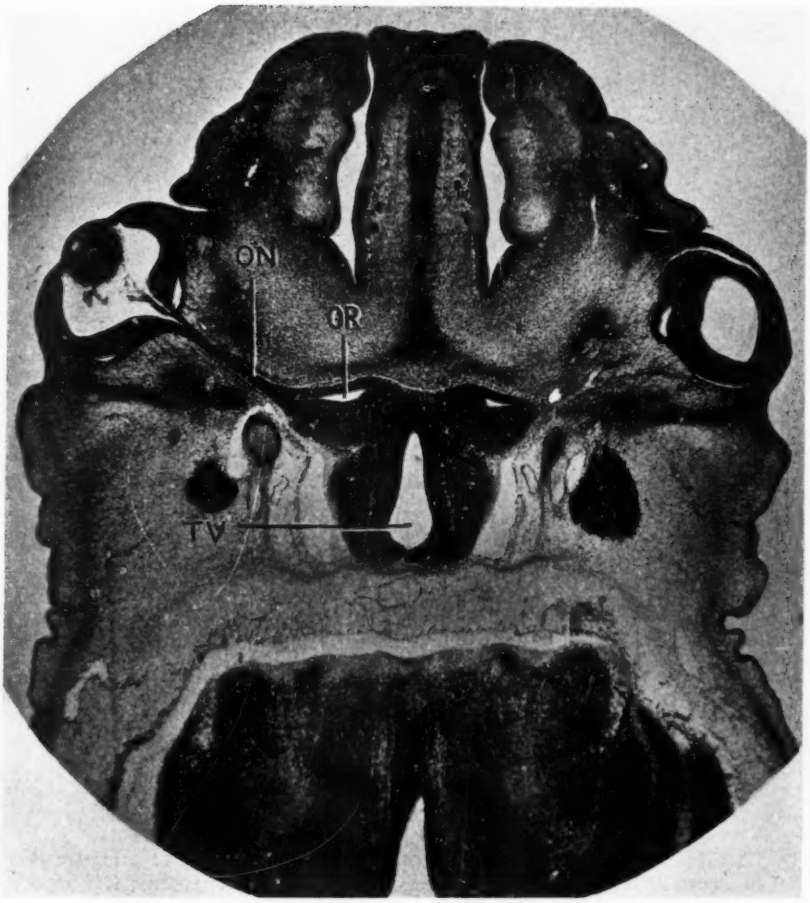

Fig. 21 (Haden). Transverse section of 24 m.m. foetus at slightly lower level but in the same plane as figure 20. Central part of OR, optic recess, has disappeared as section has passed below it. Either end of recess tapers to ON, optic nerves; TV, third ventricle.

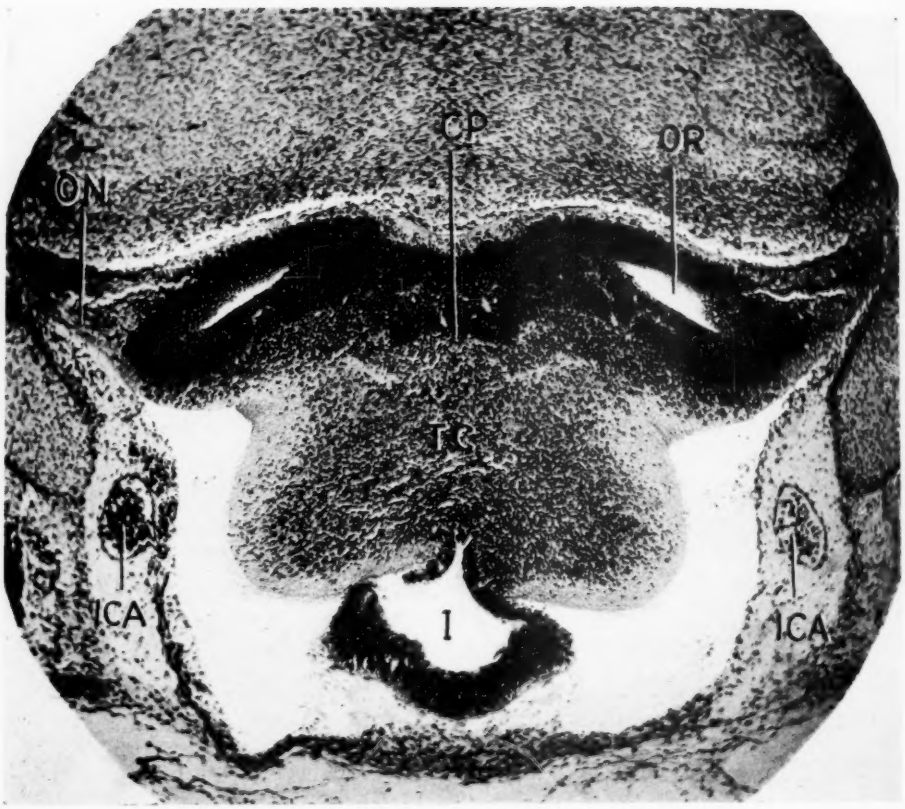

Fig. 22 (Haden). Transverse section of 24 m.m. foetus, higher magnification. Lower level than figure 21 but in the same plane. OR, lumen of optic recess; ON, optic nerve on either side; CP, chiasm primordium; TC, tuber cinereum; with opening in its caudal portion leading into I, infundibulum, note distance between primordium of optic chiasm and infundibulum; ICA, internal carotid artery.

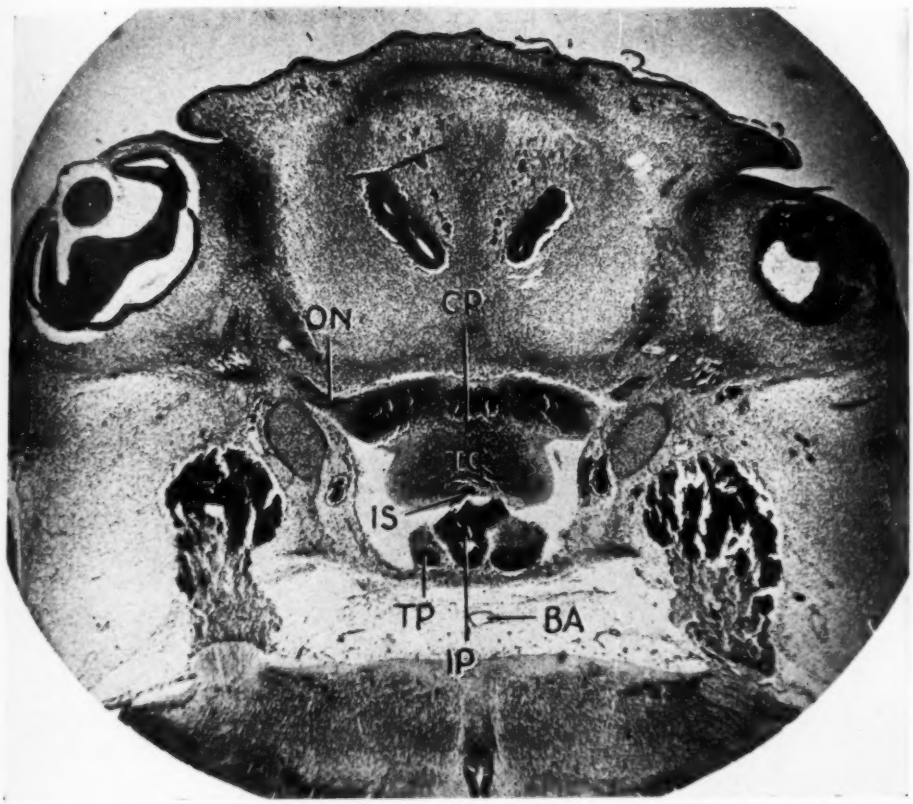

Fig. 23 (Haden). Transverse section of 24 m.m. foetus at lower level but in the same plane as figure 22. CP, chiasm primordium; ON, optic nerve; TC, tuber cinereum; IS, infundibulum stalk (stem) cut obliquely; IP, infundibular process; TP, tuberal process; BA, basilar artery.

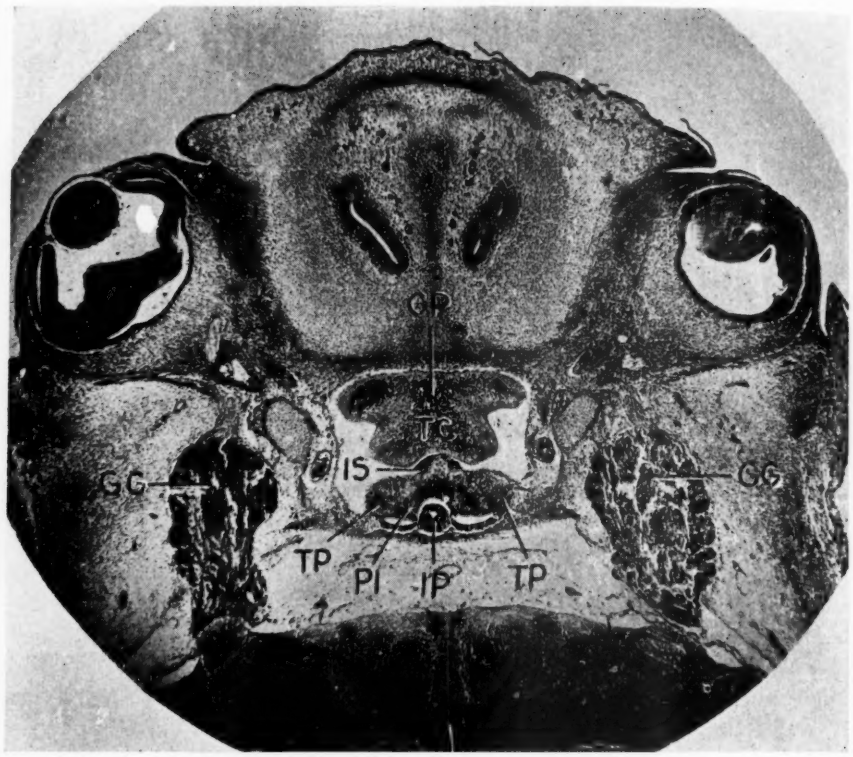

Fig. 24 (Haden). Transverse section of 24 m.m. foetus at lower level than figure 23. CP, chiasm primordium; TC, tuber cinereum; IS, infundibular stalk (stem) where cut; IP, infundibular process with central lumen; PI, pars intermedia (infundibularis); TP, tuberal processes; GG, Gasserian ganglion.

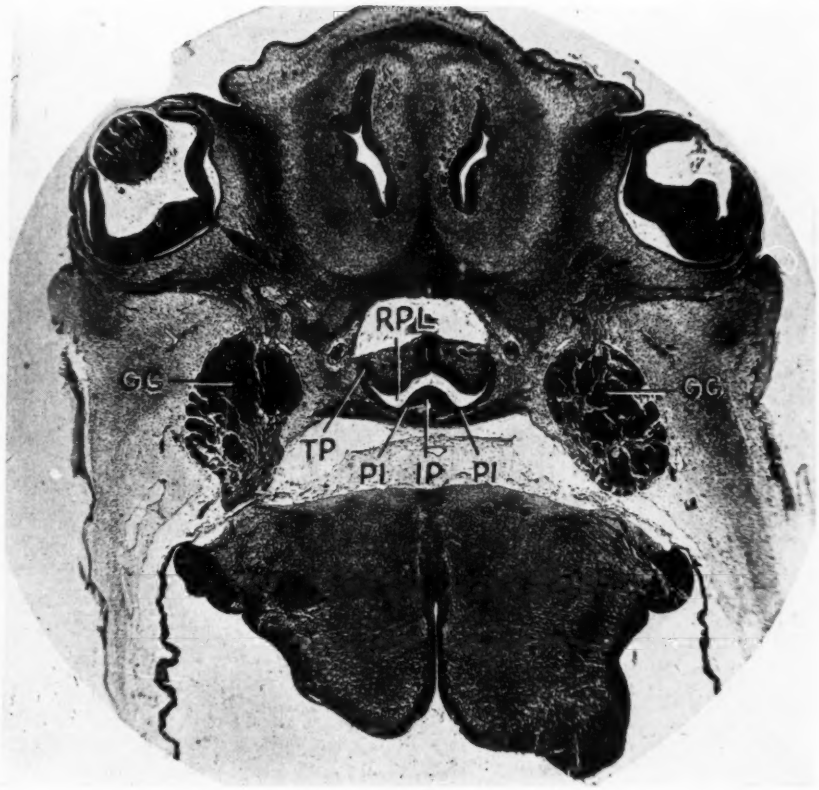

Fig. 25 (Haden). Transverse section of 24 m.m. foetus in same plane as figure 24 but at lower level. RPL, typically shaped Rathke's pouch; IP, infundibular process; PI, pars intermedia (pars infundibularis); TP, tuberal process; GG, Gasserian ganglion.

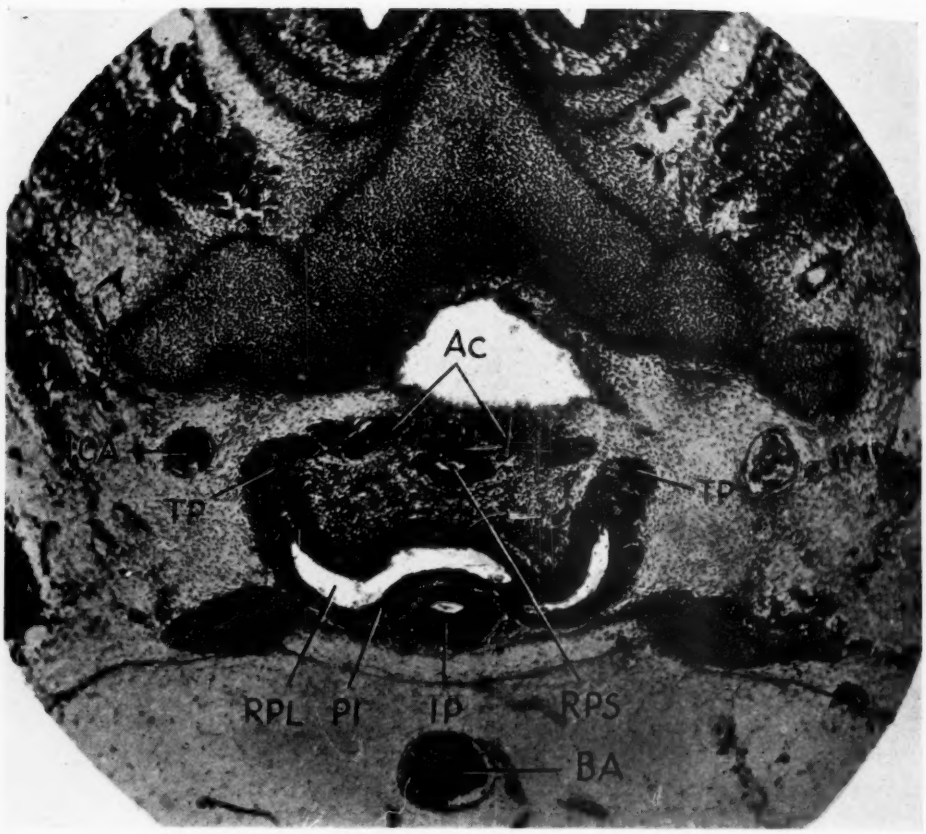

Fig. 26 (Haden). Higher magnification transverse section of 24 m.m. foetus at lower level than figure 25. Ac, acini, rosettes; RPL, Rathke's pouch lumen; RPS, Rathke's pouch stalk with lumen; TP, tuberal processes; PI, pars intermedia; IP, infundibular process with lumen; ICA, internal carotid artery; BA, basilar artery.

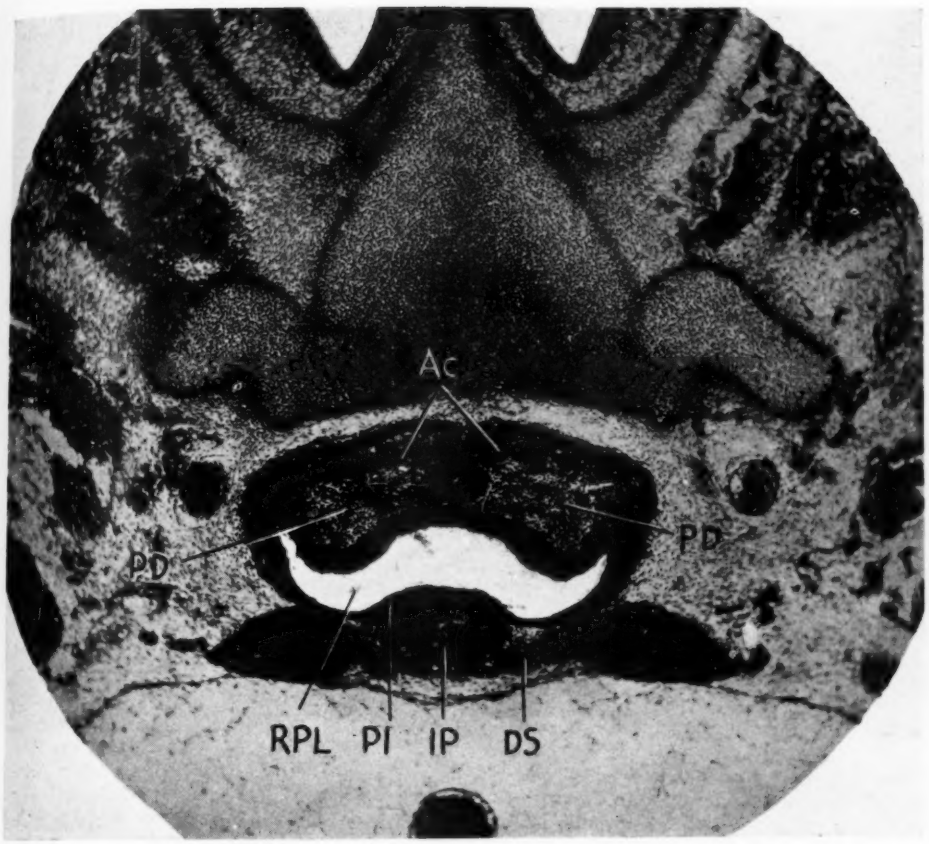

Fig. 27 (Haden). Higher magnification. Transverse section of 24 m.m. foetus at lower level than figure 26. Ac, acini connecting tuberal processes; PD, pars distalis (pars anterior propria); RPL, Rathke's pouch lumen; PI, pars intermedia; IP, infundibular process; DS, primordium of dorsum sella.

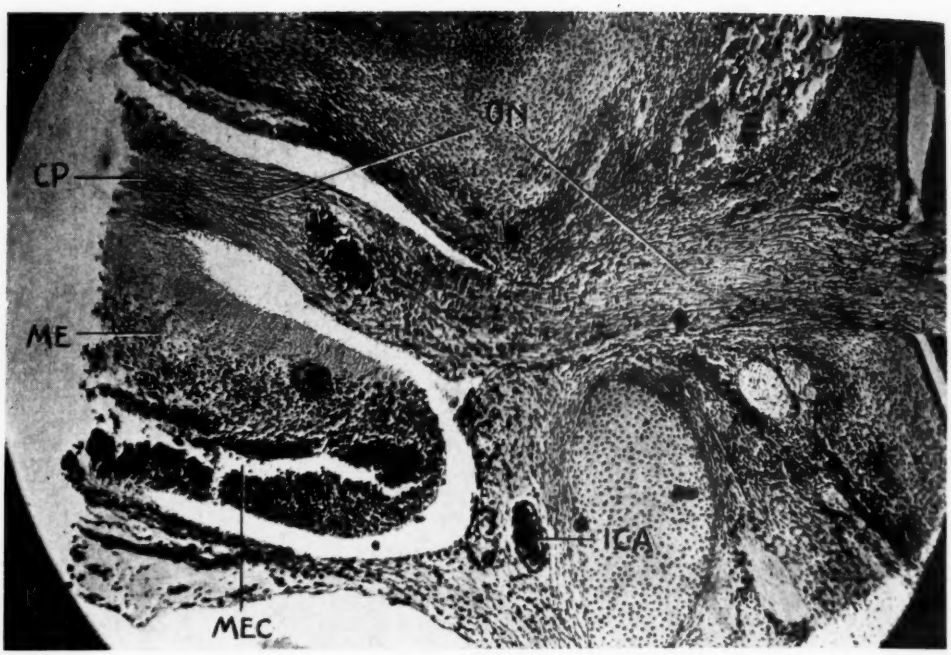

Fig. 28 (Haden). Higher magnification. Transverse section of half of head of a 29 m.m. foetus passing horizontally through the ON, optic nerve; fibers of nerve turn caudally when they reach the CP, chiasm primordium; chiasm is continuous with ME, median eminence of tuber cinereum which has a horizontally extended, MEC, cavity; ICA, internal carotid artery.

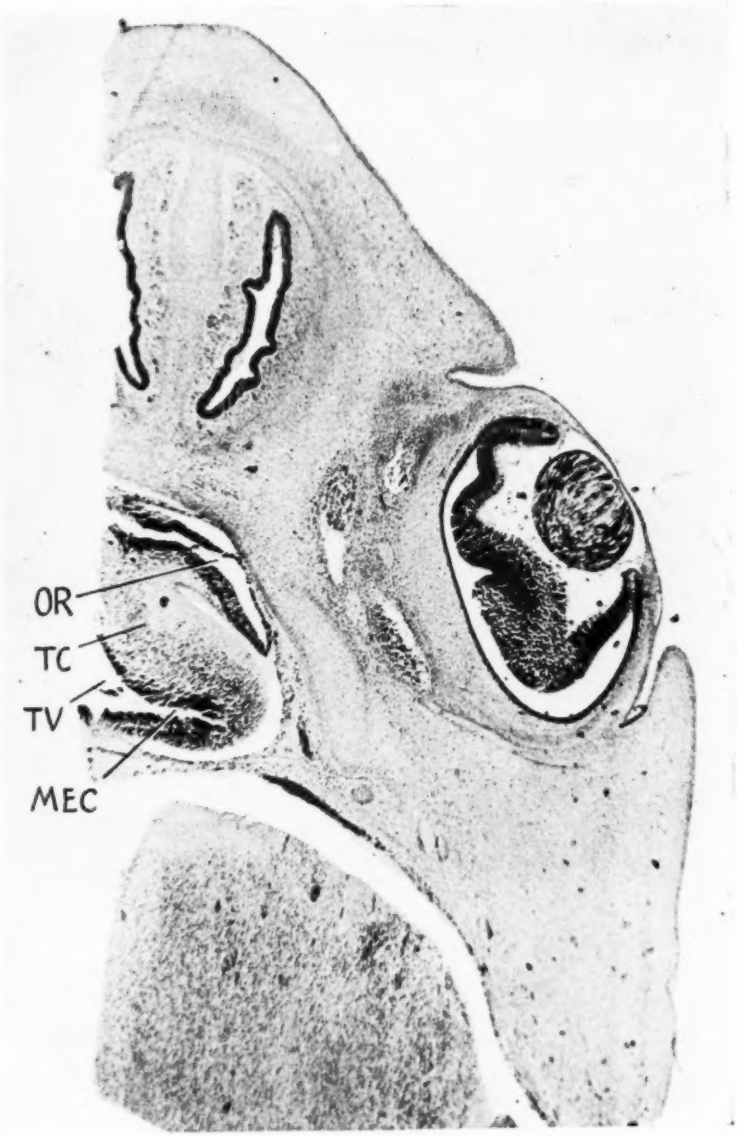

Fig. 29 (Haden). Low magnification. Transverse section of half of head of 29 m.m. foetus in same plane as figure 28 but above the level of optic nerve. OR, optic recess; TC, tuber cinereum; TV, third ventricle; MEC, median eminence cavity.

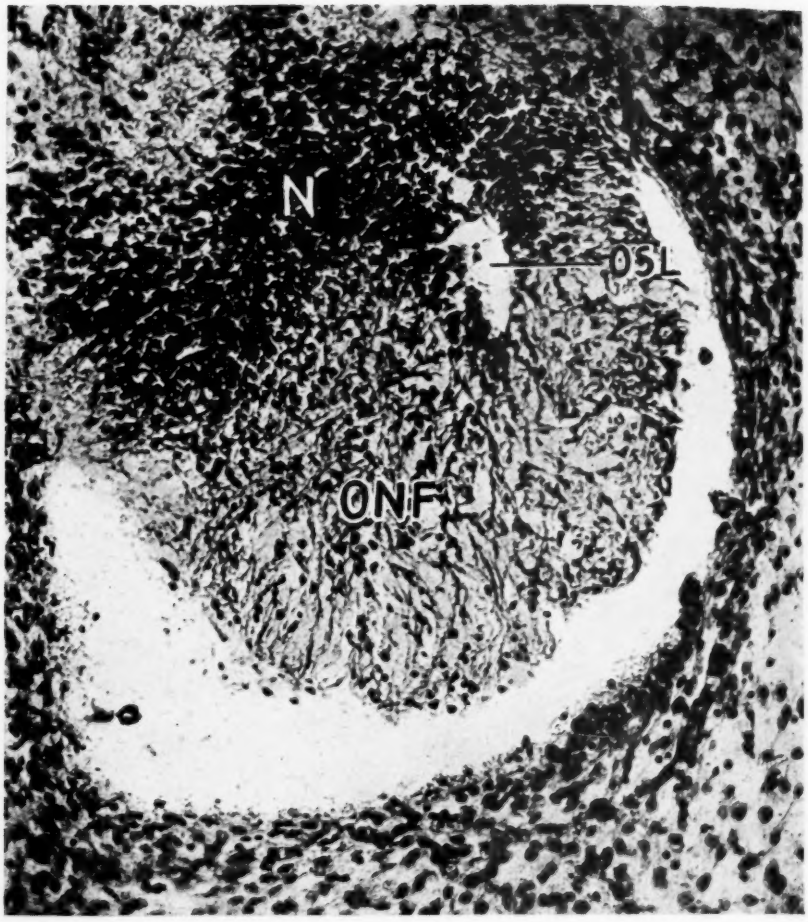

Fig. 30 (Haden). Higher magnification. Cross section of fellow nerve of 29 m.m. foetus within the cranium. N, nuclei; OSL, optic stalk lumen; ONF, optic nerve fibers.

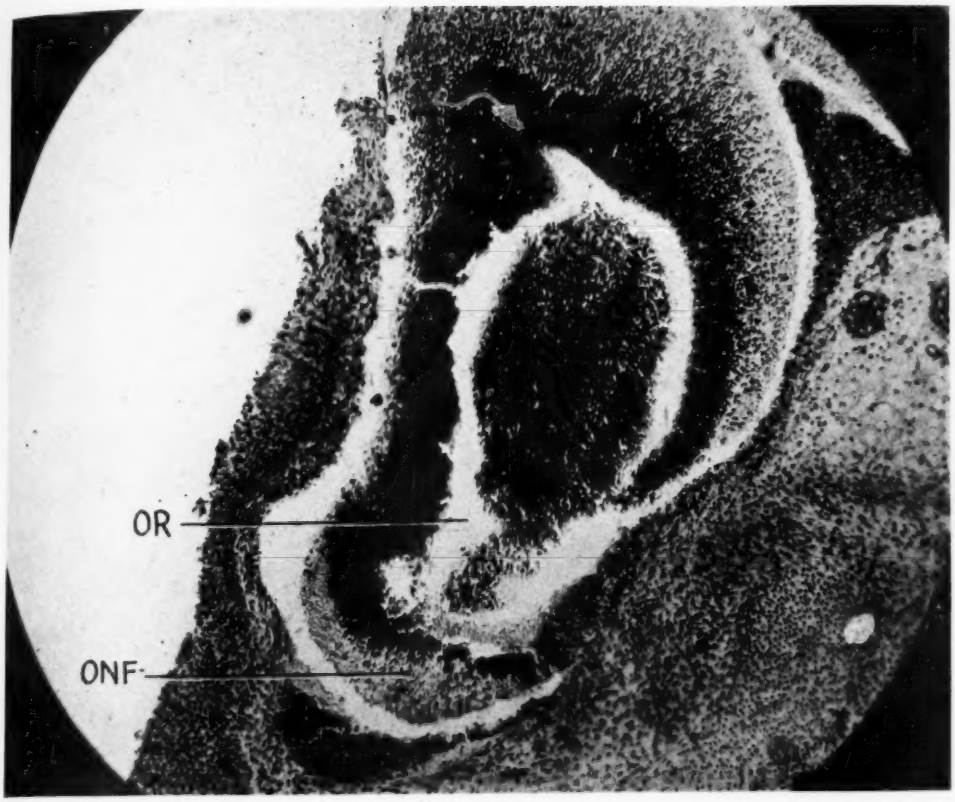

Fig. 31 (Haden). Higher magnification. 29 m.m. foetus. Cross section deeper in brain. Lumen of optic nerve goes into lumen of OR, optic recess; narrow zone of ONF, optic nerve fibers, below.

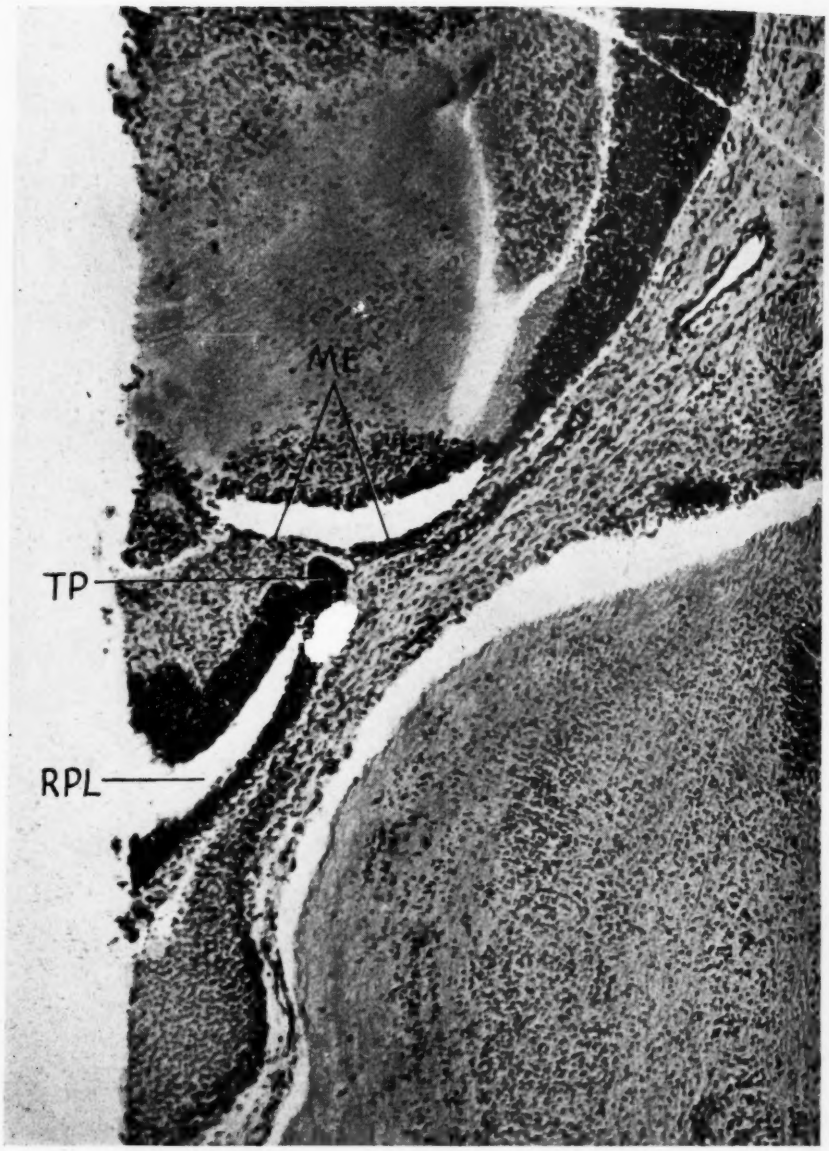

Fig. 32 (Haden). Higher magnification of 29 m.m. foetus. Cross section farther in brain; TP, tuberal process of epithelial portion of hypophysis reaching to ME, median eminence, of tuber cinereum; RPL, Rathke's pouch lumen.

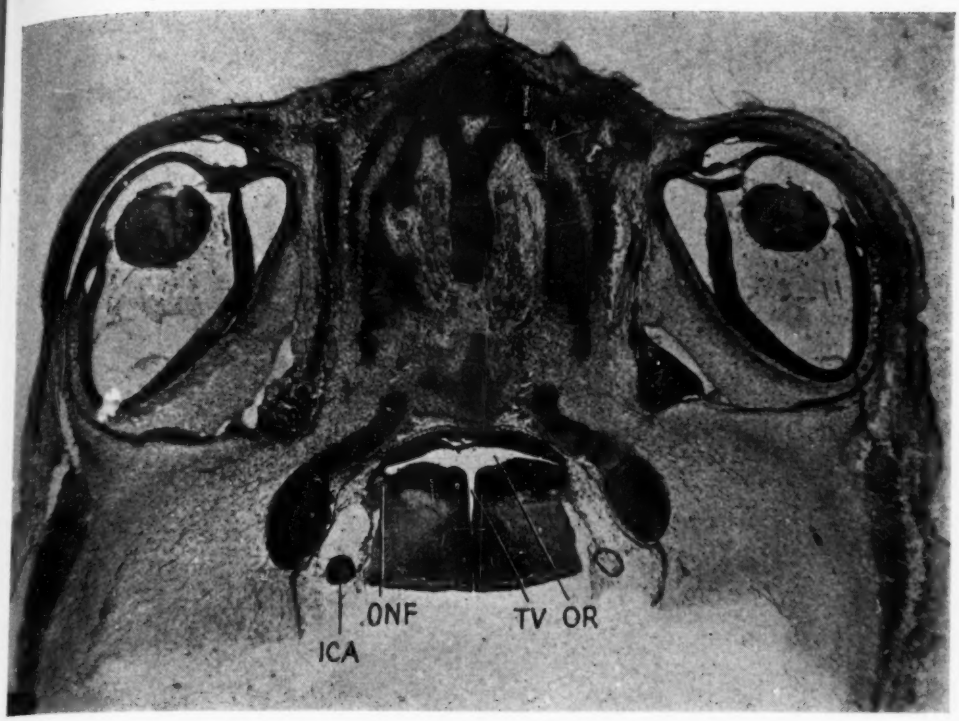

Fig. 33 (Haden). Transverse section of head of 40 m.m. foetus above level of optic nerve. OR, Optic recess communicates with TV, third ventricle, which is a narrow slitlike cavity whose walls are in contact in the middle portion; ONF, optic nerve fibers; ICA, internal carotid artery.

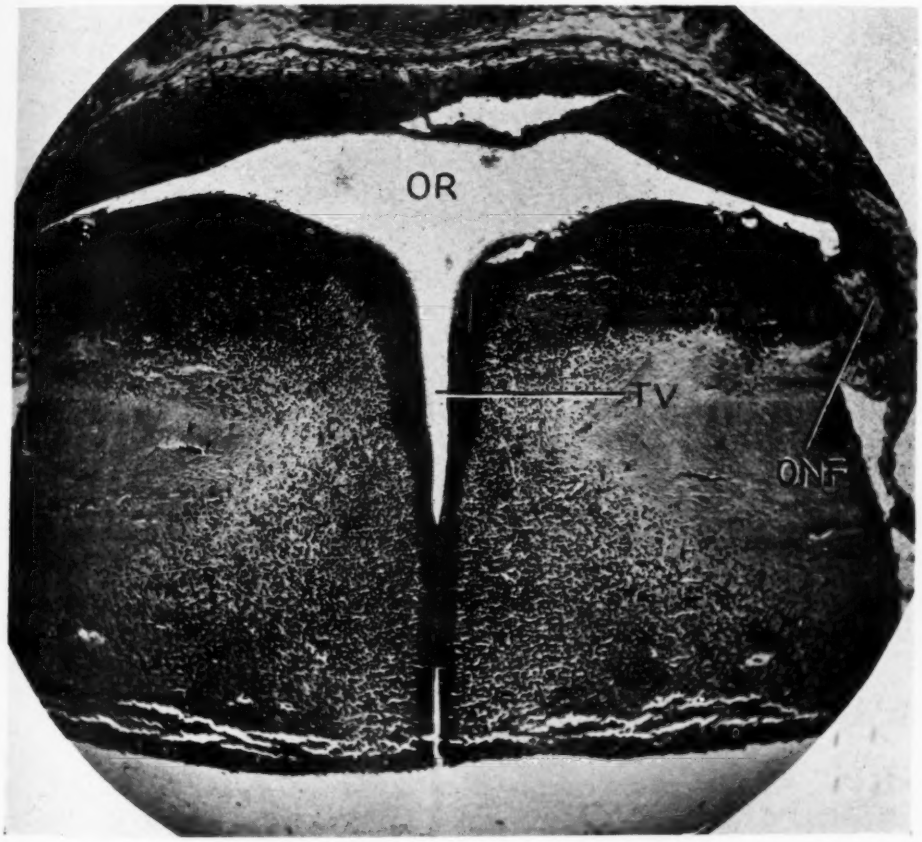

Fig. 34 (Haden). Higher magnification of figure 33. OR, optic recess; ONF, section of optic nerve; TV, third ventricle.

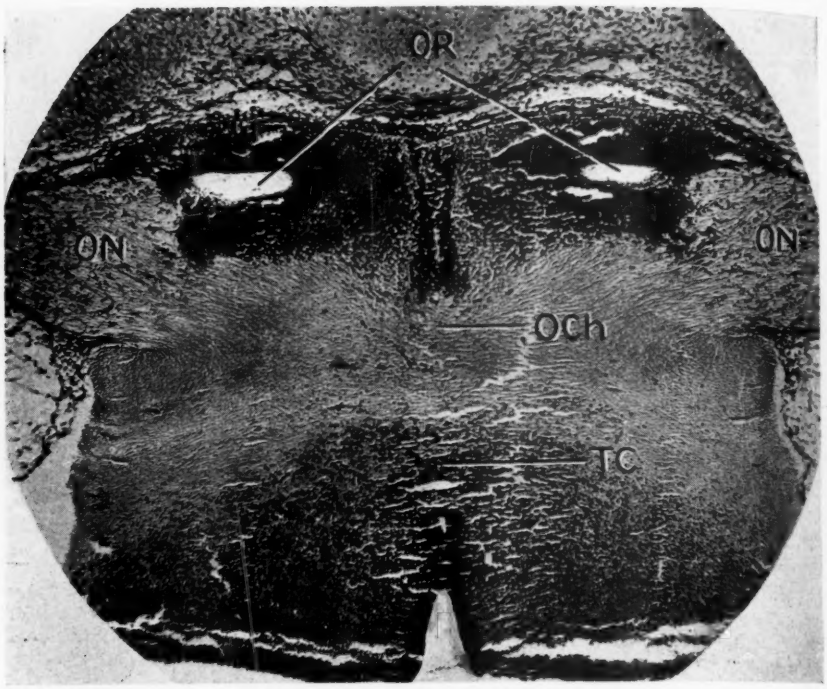

Fig. 35 (Haden). Transverse section of 40 m.m. foetus at a lower level than figure 34 but in the same plane. OR, lumen of optic recess, caudad to which ON, optic nerves, are seen entering OCh, optic chiasm; TC, tuber cinereum.

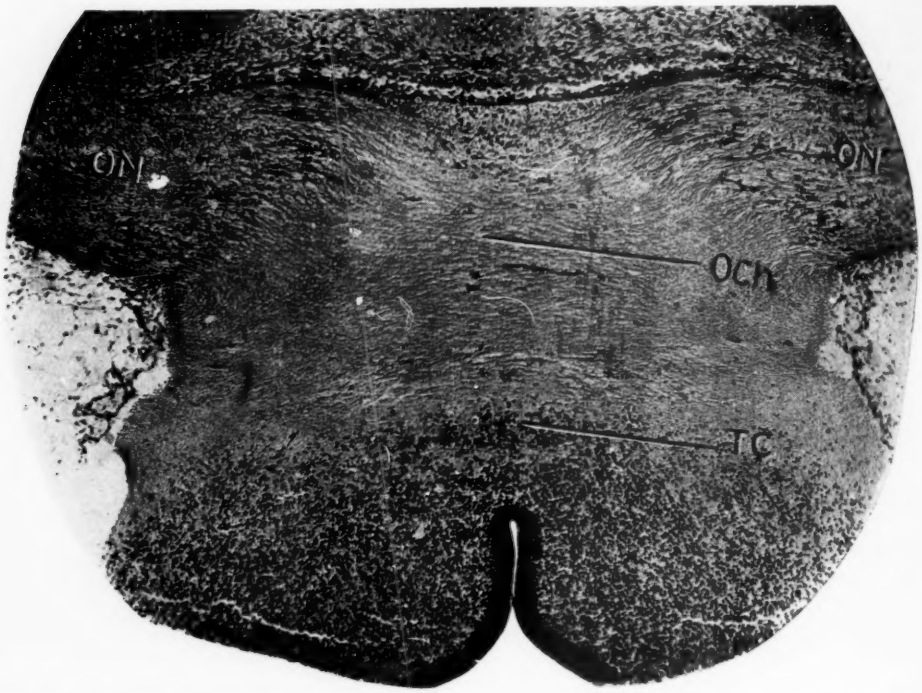

Fig. 36 (Haden). Transverse section of 40 m.m. foetus at lower level than figure 35 but in the same plane. OCh, optic chiasm, which is continuous with TC, tuber cinereum; ON, optic nerves.

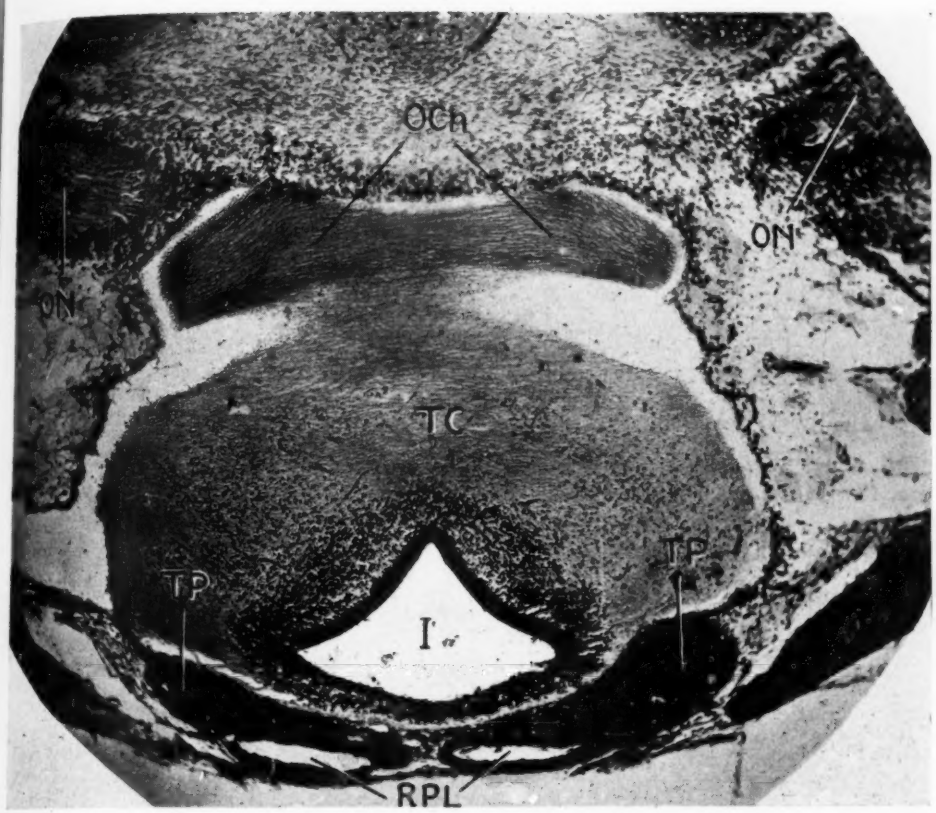

Fig. 37 (Haden). Higher magnification. Transverse section of 40 m.m. foetus at lower level than figure 36. ON, portion of optic nerves; OCh, lower part of optic chiasm; TC, tuber cinereum with opening in posterior portion leading into I, infundibulum; TP, tubera! processes of Rathke's pouch; RPL, Rathke's pouch lumen (note distance of infundibulum from chiasm).

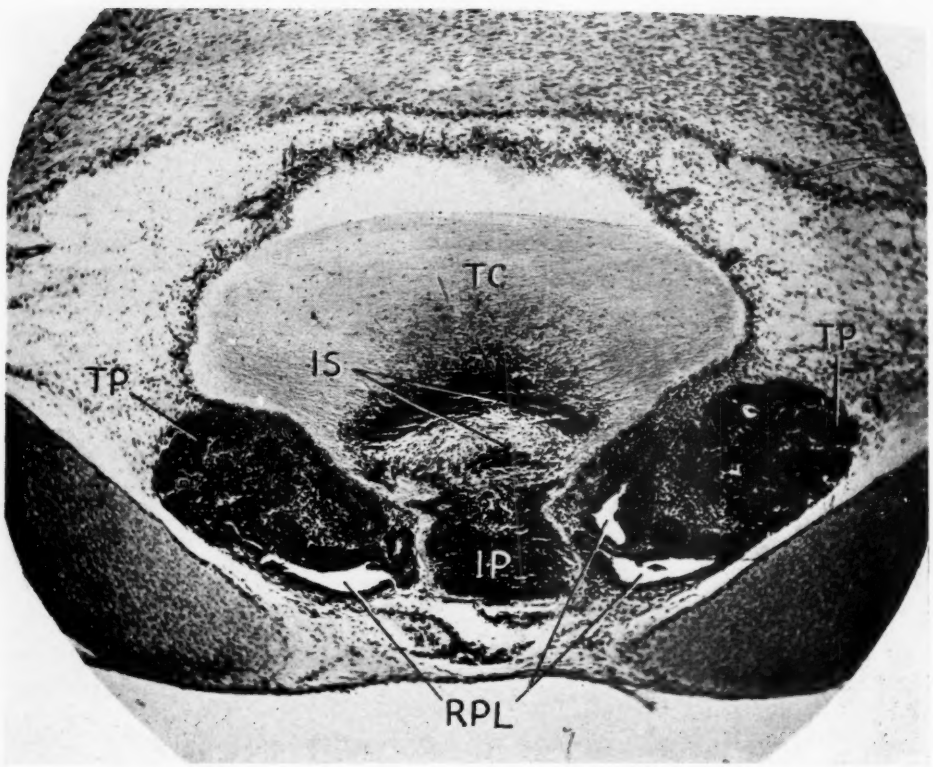

Fig. 38 (Haden). Higher magnification. Transverse section of 40 m.m. foetus at lower level than figure 37 but in the same plane. Section passes obliquely through the IS, infundibular stalk (stem), and IP, infundibular process; TP, tuberal process, filled with epithelial cells and mesenchyme; RPL, Rathke's pouch lumen; TC, tuber cinereum.

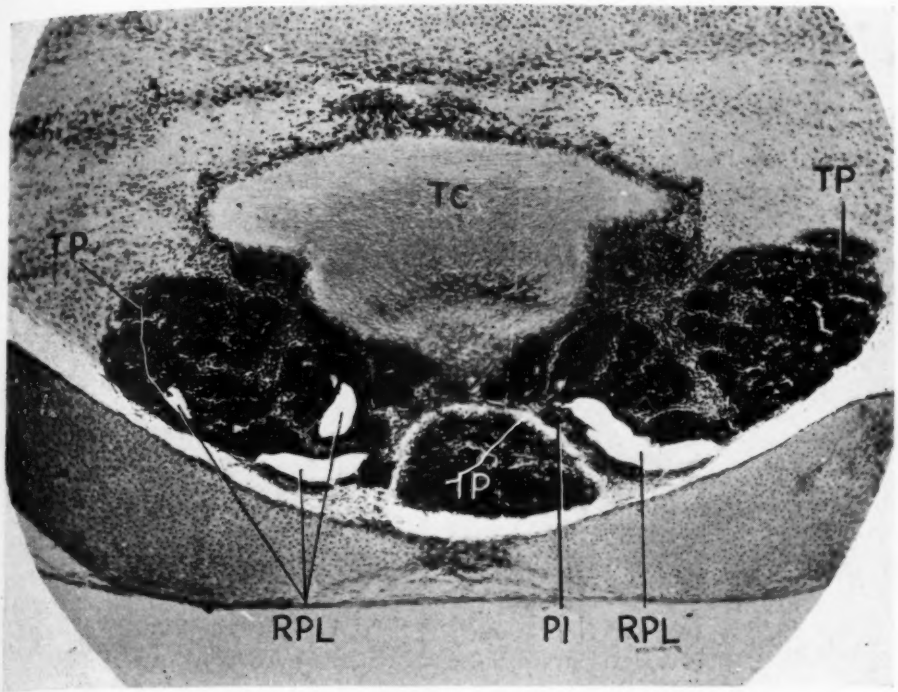

Fig. 39 (Haden). Transverse section of 40 m.m. foetus at a lower level than figure 38 but in the same plane, passing through TC, median eminence of tuber cinereum; TP, tuberal processes; RPL, Rathke's pouch lumen; PI, pars intermedia; IP, infundibular process.

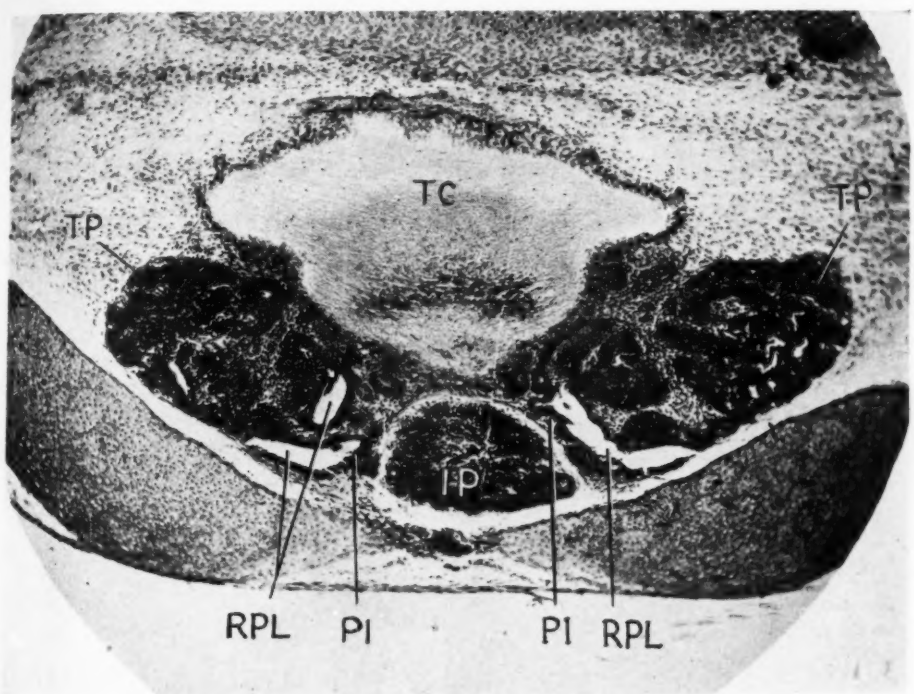

Fig. 40 (Haden). Transverse section of 40 m.m. foetus at a lower level than figure 39 but in the same plane. TC, median eminence of tuber cinereum; TP, tuberal processes; PI, pars intermedia; IP, infundibular process; RPL, Rathke's pouch lumen.

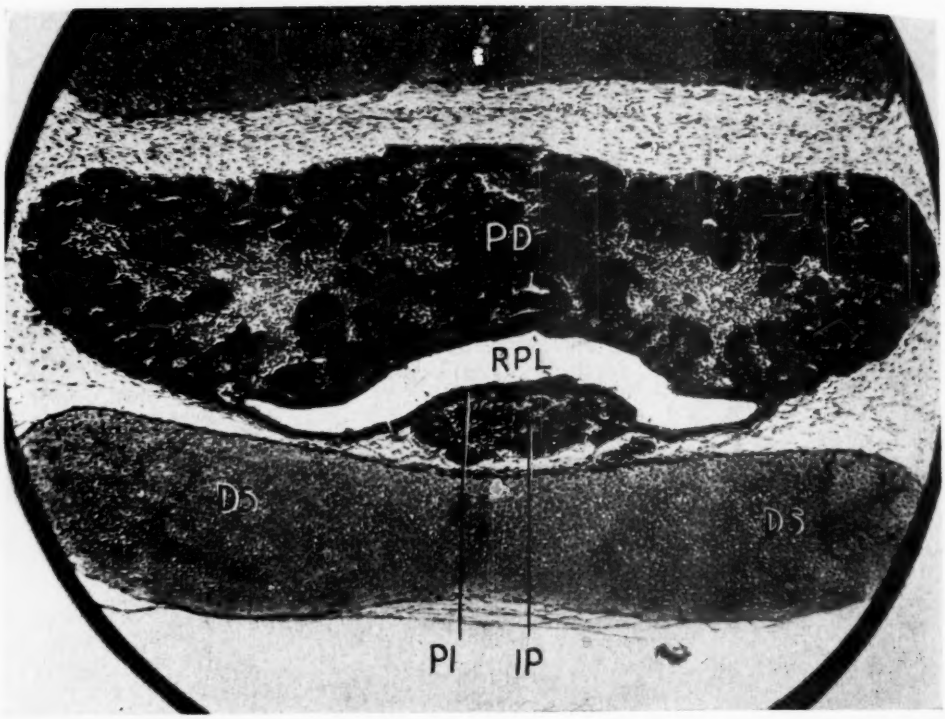

Fig. 41 (Haden). Transverse section of 40 m.m. foetus at much lower level than figure 40 but in the same plane. PD, pars distalis (pars anterior propria); RPL, Rathke's pouch lumen; PI, pars intermedia (pars infundibularis), and IP, infundibular process lying in primordium of sella turcica; DS, dorsum sella primordium.